A Survey Based Study on Awareness of Eye Diseases among the population of Bangladesh

A Dissertation submitted to the Department of Pharmacy, East West University, in partial fulfillment of the requirements for the degree of Masters of Clinical Pharmacy and Molecular Pharmacology

Submitted By

Syed Md. Nazmul Hudda

ID: 2014-01-79-005

Department of Pharmacy

East West University

January 2016

Department of Pharmacy

EAST WEST UNIVERSITY

DECLARATION BY THE CANDIDATE

I hereby declare that this dissertation, entitled "**A Survey Based Study on Awareness of Eye Diseases among the population of Bangladesh**" is an authentic and genuine research work carried out by me in partial fulfillment of the requirement for the Degree of Masters of Clinical Pharmacy and Molecular Pharmacology under the guidance of **Nishat Nasrin**, Senior lecturer, Department of Pharmacy, East West University, Dhaka.

.....

Syed Md. Nazmul Hudda

ID: 2014-1-79-005

Department of Pharmacy

East West University, Dhaka

CERTIFICATE BY THE SUPERVISOR

This is to certify that the dissertation, entitled **"A Survey Based Study on Awareness of Eye Diseases among the population of Bangladesh"** is an authentic research work done by **Syed Md. Nazmul Hudda**, ID: 2014-1-79-005, in partial fulfillment of the requirement for the Degree of Masters of Clinical Pharmacy and Molecular Pharmacology.

.....

Nishat Nasrin
Senior Lecturer
Department of Pharmacy
East West University, Dhaka

ENDORSEMENT BY THE CHAIRPERSON

"A Survey Based Study on Awareness of Eye Diseases among the population of Bangladesh" is an authentic research work done by **Syed Md. Nazmul Hudda** ID: 2014-1-79-005, in partial fulfillment of the requirement for the Degree of Masters of Clinical Pharmacy and Molecular Pharmacology under the guidance of **Nishat Nasrin**, Senior Lecturer, Department of Pharmacy, East West University, Dhaka.

.....

Dr. Shamsun Nahar Khan

Chairperson and Associate Professor

Department of Pharmacy

East West University, Dhaka

Dedicated To My Beloved Parents and Teachers

ACKNOWLEDGEMENT

First, all praise and glory are to the Almighty Allah for His mercy and blessings. I would like to express my deepest gratitude to my respected teacher and supervisor **Nishat Nasrin**, Senior Lecturer, Department of Pharmacy, East West University, for her day to day mastermind direction, constant supervision and support, constructive suggestions, optimistic counseling, valuable criticism, keen interest and active encouragement to carry out this research work.

It gives me immense pleasure to express sincere appreciation and gratefulness to **Dr. Shamsun Nahar Khan**, Chairperson and Associate Professor, Department of Pharmacy, East West University for providing all possible help during the course of this research work.

I would like to express my deepest gratitude to my respected teacher **Dr. Chowdhury Faiz Hossain,** Professor**,** Department of Pharmacy, East West University for his proper advice and support, constructive suggestions from the starting of this research work.

It is also great pleasure for me to offer my deepest indebtedness to all of my respected teachers of the Department of Pharmacy, East West University for extending their helping hands whenever needed.

Table of Contents

List of Tables

List of Figures

List of abbreviations

e.g. = *exampli gratia* = for example

Et al. = *et alia* = and other people

etc. = et cetera = and the other

Fig = Figure

i.e. = *id est.* = that is

AMD = Age related macular degeneration

ABSTRACT

Awareness of common eye diseases and their treatment can play an important role in encouraging people to seek timely eye care and can therefore help in reducing the burden of visual impairment. Some studies on awareness of eye diseases in the developed world have been carried out but such information is not sufficient and available for the Bangladeshi population, most in rural and slum population of Bangladesh. The purpose of this study was to assess the level of awareness of eye diseases in the population of Bangladesh in different location. A total of 750 people were taken under this study and a structured questionnaire was used for data collection. Among the total population 37% were female and 63% were male. From the study it was found that only 50% of the populations were aware of Glaucoma and only 30% had the knowledge about Glaucoma. This study shows that 52% of the populations were aware of Cataract and 81% people had the knowledge that risk of Cataract increases by getting older. Among the population 62% people knew the common cause of night blindness, though majority of them were urban people. From this study it was found that only 40% of the population knows that due to diabetes eye can affected. Only 23% people were concern about their family's present eye health condition and that is relatively poor. Among the population only 32% people wear sunglass for the protection of their eye. Frequency of taking nutritional food (colorful fruit, small fish, vegetables, fresh meat) for optimum eye health was very poor, only 21% take these foods daily. From this study we can see that only 36% people maintain their BP and obesity. According to this study it is very clear that awareness of eye diseases among the population of Bangladesh is not sufficient and they have not much knowledge of eye diseases.

Key words: Glaucoma, Cataract, Night blindness, Awareness, Risk factor, sight test, eye disease, eye health, sign and symptoms, treatment.

Chapter one

Introduction

1.8 Overview

Glaucoma prevalence is relatively high in Bangladesh, although it accounts for only a small proportion of blindness in the community. It is estimated that there are approximately 586 000 people 40 years and older with definite or probable glaucoma in Bangladesh (Rahman *et al*.,2004).

In Bangladesh cataract is probably responsible for over 60% of blindness, but more precise data will shortly be available as a result of a national survey. A cataract surgical rate of at least 2000 cataract operations / year / million populations is needed to control cataract blindness effectively. However, the present performance is estimated to be less than 500 per annum. With a population of 130 million (which is growing at the rate of 2.1%/year), a minimum of 260,000 cataract operations / year are needed in Bangladesh (National Institute of Ophthalmology, 1999).

Most eye care services, which are concentrated in urban areas, are clinically orientated and provide curative services. The majority does not have regular outreach activities, nor do they undertake any preventive work. There are approximately 400 qualified ophthalmologists in the country, and a further 700 who have been trained in ophthalmology, but the level of service provision is inadequate to meet the need, mainly due to misdistribution of resources. Comprehensive eye care services, which encompass prevention, health promotion, rehabilitation as well as curative, clinical services are mostly unavailable.

Surgical eye camps are very popular with Bangladeshi people, because the services are usually provided free. However, eye camps are undertaken less frequently now, due to advances in surgical techniques, and awareness of the need for high quality surgery and good post-operative follow up. In addition, there are questions of social equity and justice to be considered (Foster A, 1999).

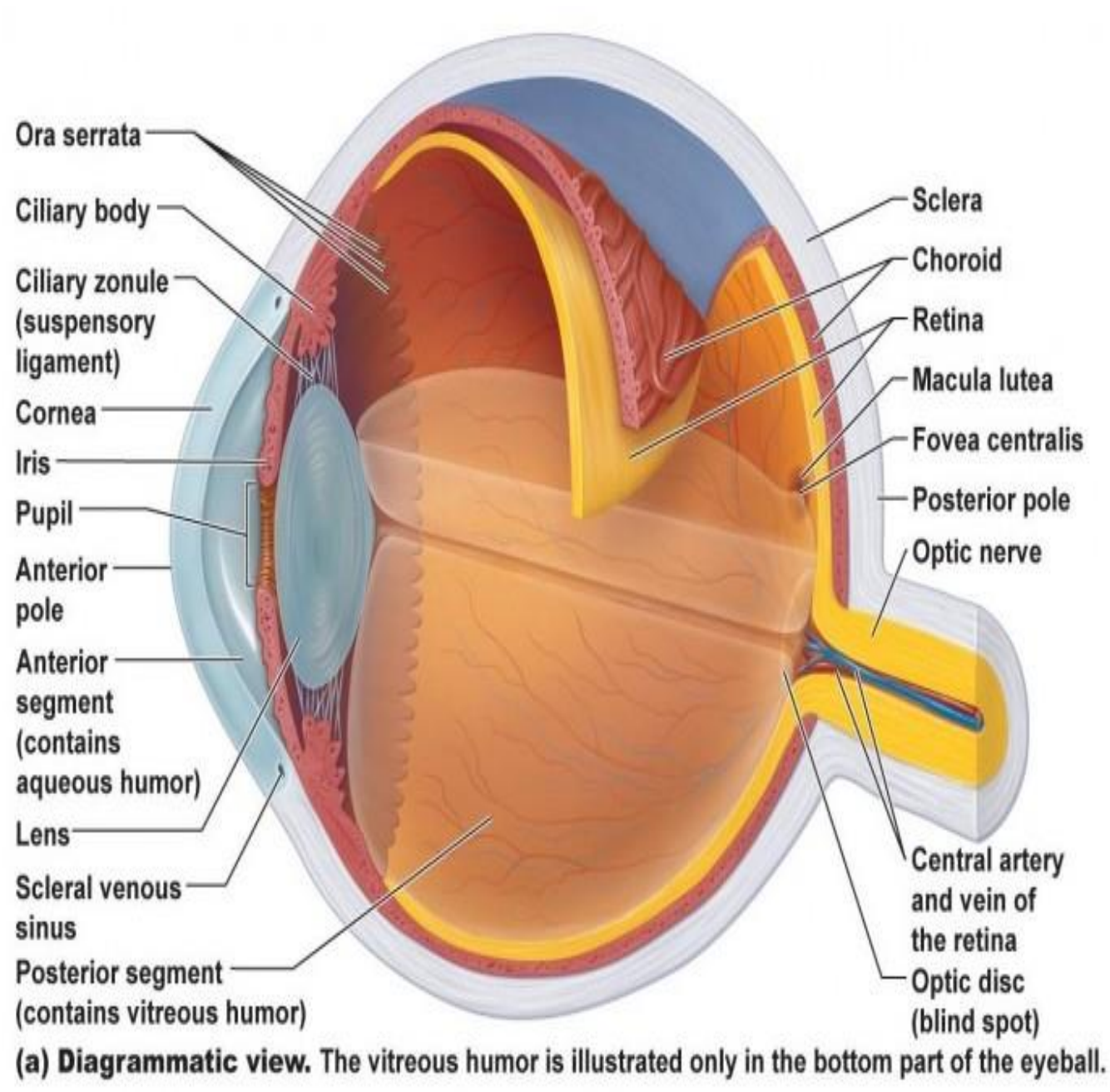


Figure 1.1: Anatomy of human eye (source: Pearson education Inc., 2011, http://m.wikitree.co.kr/main/news_view.php?id=231044)

1.9 Common Eye Disorders

Approximately 11 million Americans aged 12 years and older could improve their vision through proper refractive correction. More than 3.3 million Americans aged 40 years and older are either legally blind (having best-corrected visual acuity of 6/60 or worse (=20/200) in the better-seeing eye) or are with low vision (having best-corrected visual acuity less than 6/12 (<20/40) in the better-seeing eye, excluding those who were categorized as being blind). The leading causes of blindness and low vision in the United States are primarily age-related eye diseases such as age-related macular degeneration, cataract, diabetic retinopathy, and glaucoma. Other common eye disorders include amblyopia and strabismus (Rhee *et al*., 2013).

1.10 Glaucoma

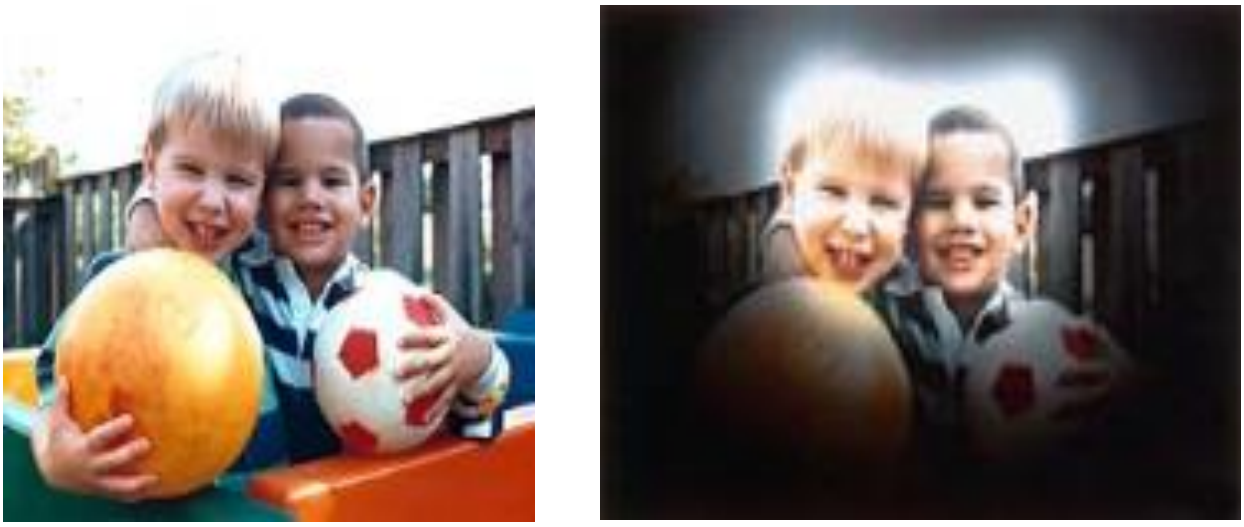
Glaucoma is a term for a group of eye disorders which result in damage to the optic nerve. This is most often due to increased. The disorders can be roughly divided into two main categories: "open-angle" and "closed-angle" (or "angle closure") glaucoma. Open-angle chronic glaucoma is painless, tends to develop slowly over time and often has no symptoms until the disease has progressed significantly. It is treated with either glaucoma medication to lower the pressure, or with various pressure-reducing glaucoma surgeries. Closed-angle glaucoma, however, is characterized by sudden eye pain, redness, nausea and vomiting, and other symptoms resulting from a sudden spike in intraocular pressure, and is treated as a medical emergency. Glaucoma can permanently damage vision in the affected eye(s), first by decreasing peripheral vision (reducing the visual field), and then potentially leading to blindness if left untreated (Xue-Song *et al*., 2014).

The many different subtypes of glaucoma can all be considered to be a type of optic neuropathy. The nerve damage involves loss of retinal ganglion cells in a characteristic pattern. Raised intraocular pressure (above 21 mmHg or 2.8 kPa) is the most important and only modifiable risk factor for glaucoma. Some may have high eye pressure for years and never develop damage, a condition known as "ocular hypertension". Conversely, the term 'low

tension' or 'normal tension' glaucoma is used for those with optic nerve damage and associated visual field loss, but normal or low intraocular pressure (Leffler *et al*., 2013).

Glaucoma has been called the "silent thief of sight" because the loss of vision often occurs gradually over a long period of time, and symptoms only occur when the disease is quite advanced. Worldwide, glaucoma is the second-leading cause of blindness after cataracts. It is also the leading cause of blindness among African Americans.

If the condition is detected early enough, it is possible to arrest the development or slow the progression with medical and surgical means. Although the term "glaucoma" has a history relating to disorders of the eye going back to ancient Greece, in English the word was not commonly used until after 1850, when the development of the ophthalmoscope permitted visualization of the optic nerve damage caused by glaucoma (Sommer *et al*., 1991).



Normal vision vision loss

Figure 1.2: vision loss from glaucoma (source: National eye institute, http://www.nei.nih.gov/photo/keyword.asp?narrow=Eye+Disease+Simulation&match=all)

1.10.1 Signs and symptoms

Open-angle glaucoma is painless and does not have acute attacks, thus the lack of clear symptoms make screening via regular eye checkups important. The only signs are gradually progressive visual field loss, and optic nerve changes (increased cup-to-disc ratio on fundoscopic examination).

About 10% of people with closed-angles present with acute angle closure characterized by sudden ocular pain, seeing halos around lights, red eye, very high intraocular pressure (>30 mmHg), nausea and vomiting, suddenly decreased vision, and a fixed, mid-dilated pupil. It is also associated with an oval pupil in some cases. Acute angle closure is an emergency.

1.10.2 Causes

Of the several causes for glaucoma, ocular hypertension (increased pressure within the eye) is the most important risk factor in most glaucoma, but in some populations, only 50% of people with primary open-angle glaucoma actually have elevated ocular pressure (Li *et al*., 2011).

Open-angle glaucoma accounts for 90% of glaucoma cases in the United States. Closed-angle glaucoma accounts for less than 10% of glaucoma cases in the United States, but as many as half of glaucoma cases in other nations (particularly Asian countries).

Dietary

No clear evidence indicates vitamin deficiencies cause glaucoma in humans. It follows, then, that oral vitamin supplementation is not a recommended treatment for glaucoma. Caffeine increases intraocular pressure in those with glaucoma, but do not appear to affect normal individuals (Wang *et al*., 2002).

Ethnicity and sex

Many people of East Asian descent are prone to developing angle closure glaucoma due to shallower anterior chamber depths, with the majority of cases of glaucoma in this population consisting of some form of angle closure. Inuit also have a 20- to 40-times higher risk of developing primary angle closure glaucoma. Women are three times more likely than men to develop acute angle closure glaucoma due to their shallower anterior chambers. People of African descent are three times more likely to develop primary open-angle glaucoma (Chang *et al*., 2001).

Genetics

Positive family history is a risk factor for glaucoma. The relative risk of having primary open-angle glaucoma (POAG) is increased about two- to four-fold for individuals who have a sibling with glaucoma. Glaucoma, particularly primary open-angle glaucoma, is associated with mutations in several different genes (including MYOC, ASB10, WDR36, NTF4, and TBK1genes), although most cases of glaucoma do not involve these genetic mutations. Normal-tension glaucoma, which comprises one-third of POAG, is also associated with genetic mutations (including OPA1 and OPTN genes).

Various rare congenital/genetic eye malformations are associated with glaucoma. Occasionally, failure of the normal third-trimester gestational atrophy of the hyaloids canal and the tunica vasculosa lentis is associated with other anomalies. Angle closure-induced ocular hypertension and glaucomatous optic neuropathy may also occur with these anomalies, and has been modeled in mice (Quigley *et al*., 2006).

Other

Other factors can cause glaucoma, known as "secondary glaucoma", including prolonged use of steroids (steroid-induced glaucoma); conditions that severely restrict blood flow to the eye, such as severe diabetic retinopathy and central retinal vein occlusion (neovascular glaucoma); ocular trauma (angle-recession glaucoma); anduveitis (uveitic glaucoma).

1.10.3 Epidemiology

As of 2010, there were 44.7 million people in the world with open angle glaucoma. The same year, there were 2.8 million people in the United States with open angle glaucoma. By 2020, the prevalence is projected to increase to 58.6 million worldwide and 3.4 million the United States (Ramulu *et al*., 2009).

Internationally, glaucoma is the second-leading cause of blindness, after cataracts. Glaucoma is also the leading cause of blindness in African Americans, who have higher rates of primary open angle glaucoma. Bilateral vision loss can negatively affect mobility and interfere with driving.

A meta-analysis published in 2009 found that people with primary open angle glaucoma do not have increased mortality rates, or increased risk of cardiovascular death (Akbari *et al*., 2009).

1.10.4 Pathophysiology

The underlying cause of open-angle glaucoma remains unclear. Several theories exist on its exact etiology. However, the major risk factor for most glaucomas, and the focus of treatment, is increased intraocular pressure, i.e. ocular hypertension. Intraocular pressure is a function of production of liquid aqueous humor by the ciliary processes of the eye, and its drainage through the trabecular meshwork. Aqueous humor flows from the ciliary processes into the posterior chamber, bounded posteriorly by the lens and the zonules of Zinn, and anteriorly by the iris.

It then flows through the pupil of the iris into theanterior chamber, bounded posteriorly by the iris and anteriorly by the cornea. From here, the trabecular meshwork drains aqueous humor via Schlemm's canal into scleral plexuses and general blood circulation.

In open/wide-angle glaucoma, flow is reduced through the trabecular meshwork, due to the degeneration and obstruction of the trabecular meshwork, whose original function is to absorb the aqueous humor. Loss of aqueous humor absorption leads to increased resistance and thus a chronic, painless buildup of pressure in the eye (Osborne *et al*., 1999).

In close/narrow-angle, the iridocorneal angle is completely closed because of forward displacement of the final roll and root of the iris against the cornea, resulting in the inability of the aqueous fluid to flow from the posterior to the anterior chamber and then out of the trabecular network. This accumulation of aqueous humor causes an acute increase of pressure and pain. The inconsistent relationship of glaucomatous optic neuropathy with ocular hypertension has provoked hypotheses and studies on anatomic structure, eye development, nerve compression trauma, optic nerve blood flow, excitatory neurotransmitter, trophic factor, and retinal ganglion cell/axon degeneration, and glial support cell, immune system, aging mechanisms of neuron loss, and severing of the nerve fibers at the scleral edge (Arthur *et al*., 2009).

1.10.5 Types

Glaucoma is an umbrella term for eye conditions which damage the the optic nerve, and which can lead to a loss of vision. The main cause of damage to the optic nerve is intraocular pressure (IOP), excessive fluid pressure within the eye, which can be due to various reasons including blockage of drainage ducts, and narrowing or closure of the angle between the iris and cornea.

The primary division in categorizing different types of glaucoma is open-angle and closed angle (or angle-closure) glaucoma. The open angle refers to the angle where the iris meets the cornea being as wide and open as it should be, allowing the fluid from inside the eye to drain, thus relieving the internal pressure. Where this angle is narrowed or closed, pressure can build up, and eventually damage the optic nerve leading to loss of vision (Chang *et al*., 2009).

Primary open-angle glaucoma

Refers to slow clogging of the drainage canals resulting in increased eye pressure which causes progressive optic nerve damage. This manifests as a gradual loss of the visual field, starting with a loss of peripheral vision, but eventually the entire vision will be lost if not treated. This is the most common type of glaucoma, accounting for 90% of cases in the United States, but fewer in Asian countries. Onset is slow and painless, and loss of vision is gradual and irreversible.

Narrow angle glaucoma

The iris bows forward, narrowing the angle that drains the eye, increasing pressure within the eye. If untreated, it can lead to the medical emergency of angle closure glaucoma.

Angle closure glaucoma

The iris bows forward and causes physical contact between the iris and trabecular meshwork, which in turn blocks outflow of the aqueous humor from within the eye. This contact may gradually damage the draining function of the meshwork until it fails to keep pace with aqueous production, and the intraocular pressure rises. Onset of symptoms is sudden, and causes pain and other symptoms that are noticeable, and is treated as a medical emergency. Unlike open-angle glaucoma, angle-closure glaucoma is a result of the angle between the iris and cornea closing. Tends to occur in the far-sighted, who have smaller than normal anterior chambers, making the physical contact more likely.

Normal tension glaucoma

Is a condition where the optic nerve is damaged although intraocular pressure (IOP) is in normal range (12-22mm Hg). At higher risk are those with family history of NTG, those of Japenese ancestry, and those with history of systemic heart disease. The cause of NTG is unknown.

Secondary glaucoma

Refers to any case in which another disease, trauma, drug or procedure causes increased eye pressure, resulting in optic nerve damage and vision loss, and may be mild or severe. It can be due to eye injury, inflammation, a tumor, or advanced cases of cataracts or diabetes. It can also be caused by certain drugs such as steroids. Treatment depends on whether it is open-angle or angle-closure glaucoma.

Pseudo exfoliation glaucoma

the pressure is due to the accumulation of microscopic granular protein fibers, which can block normal drainage of the aqueous humor. PEX is prevalent in Scandinavia, primarily in those over 70, and more in women.

Pigmentary glaucoma

Is caused by pigment cells sloughing off from the back of the iris and floating around in the aqueous humor. Over time, these pigment cells can accumulate in the anterior chamber in such a way that it can begin to clog the trabecular meshwork. A rare condition, it occurs mostly among Caucasians, mostly males in their mid-20s to 40s, most nearsighted.

Primary juvenile glaucoma

Is a neonate or juvenile abnormality where ocular hypertension is evident at birth or shortly thereafter and is caused by abnormalities in the anterior chamber angle development that blocks outflow of the aqueous humor.

Uveitic Glaucoma

Is due to uveitis, the swelling and inflammation of the uvea, the middle layer of the eye. The uvea provides most of the blood supply to the retina. Increased eye pressure in uveitis can result from the inflammation itself or from the steroids used to treat it.

1.10.6 Diagnosis

Screening for glaucoma is usually performed as part of a standard eye examination performed by optometrists and ophthalmologists. Testing for glaucoma should include measurements of the intraocular pressure via tonometry, anterior chamber angle examination or gonioscopy, and examination of the optic nerve to look for any visible damage to it, or change in the cup-to-disc ratio and also rim appearance and vascular change (Pardianto, 2006).

A formal visual field test should be performed. The retinal nerve fiber layer can be assessed with imaging techniques such as optical coherence tomography, scanning laser polarimetry, and/or scanning laser ophthalmoscopy.

Owing to the sensitivity of all methods of tonometry to corneal thickness, methods such as Goldman tonometry should be augmented with pachymetry to measure central corneal thickness (CCT).

A thicker-than-average cornea can result in a pressure reading higher than the 'true' pressure, whereas a thinner-than-average cornea can produce a pressure reading lower than the 'true' pressure. Because pressure measurement error can be caused by more than just CCT (i.e., corneal hydration, elastic properties, etc.), it is impossible to 'adjust' pressure measurements based only on CCT measurements.

The frequency doubling illusion can also be used to detect glaucoma with the use of a frequency doubling technology perimeter. Examination for glaucoma also could be assessed with more attention given to sex, race, history of drug use, refraction, inheritance and family history (Johnson *et al*., 2009).

Table 1.1: Glaucoma test

	What test examines	How it is accomplished
Tonometry	Inner eye pressure	The eye is numbed via eye drops. The examiner then uses a tonometer to measure the inner pressure of the eye through pressure applied by a puff of warm air or a tiny tool.
Ophthalmoscopy (dilated eye exam)	Shape and color of the optic nerve	The pupil is dilated via the application of eye drops. Using a small magnification device with a light on the end, the examiner can examine the magnified optic nerve.
Perimetry (visual field test)	Complete field of vision	The patient looks straight ahead and is asked to indicate when light passes the patient's peripheral field of vision. This allows the examiner to map the patient's field of vision.
Gonioscopy	Angle in the eye where the iris meets the cornea	Eye drops are used to numb the eye. A hand-held contact lens with a mirror is placed gently on the eye to allow the examiner to see the angle between the cornea and the iris.
Pachymetry	Thickness of the cornea	The examiner places a pachymeter gently on the front of the eye to measure its thickness.
Nerve fiber analysis	Thickness of the nerve fiber layer	Using one of several techniques, the nerve fibers are examined.

Table 1.1: Glaucoma test (source: https://en.wikipedia.org/wiki/Glaucoma)

1.10.7 Treatment

The modern goals of glaucoma management are to avoid glaucomatous damage and nerve damage, and preserve visual field and total quality of life for patients, with minimal side effects. This requires appropriate diagnostic techniques and follow-up examinations, and judicious selection of treatments for the individual patient. Although intraocular pressure is only one of the major risk factors for glaucoma, lowering it via various pharmaceuticals and/or surgical techniques is currently the mainstay of glaucoma treatment.

Vascular flow and neurodegenerative theories of glaucomatous optic neuropathy have prompted studies on various neuroprotective therapeutic strategies, including nutritional compounds, some of which may be regarded by clinicians as safe for use now, while others are on trial (Leffler *et al*., 2009).

1.10.7.1 Medication

Intraocular pressure can be lowered with medication, usually eye drops. Several different classes of medications are used to treat glaucoma, with several different medications in each class.

Each of these medicines may have local and systemic side effects. Adherence to medication protocol can be confusing and expensive; if side effects occur, the patient must be willing either to tolerate them, or to communicate with the treating physician to improve the drug regimen. Initially, glaucoma drops may reasonably be started in either one or in both eyes.

Poor compliance with medications and follow-up visits is a major reason for vision loss in glaucoma patients. A 2003 study of patients in an HMO found half failed to fill their prescriptions the first time, and one-fourth failed to refill their prescriptions a second time. Patient education and communication must be ongoing to sustain successful treatment plans for this lifelong disease with no early symptoms (Tsai *et al*,. 2007).

The possible neuroprotective effects of various topical and systemic medications are also being investigated (Pardianto, 2006).

- Prostaglandin analogs, such as latanoprost, bimatoprost and travoprost, increase uveoscleral outflow of aqueous humor. Bimatoprost also increases trabecular outflow.
- Topical beta-adrenergic receptor antagonists, such as timolol, levobunolol, and betaxolol, decrease aqueous humor production by the epithelium of the ciliary body.
- Alpha2-adrenergic agonists, such as brimonidine and apraclonidine, work by a dual mechanism, decreasing aqueous humor production and increasing uveoscleral outflow.
- Less-selective alpha agonists, such as epinephrine, decrease aqueous humor production through vasoconstriction of ciliary body blood vessels, useful only in open-angle glaucoma. Epinephrine's mydriatic effect, however, renders it unsuitable for closed-angle glaucoma due to further narrowing of the uveoscleral outflow (i.e. further closure of trabecular meshwork, which is responsible for absorption of aqueous humor).
- Miotic agents (parasympathomimetics), such as pilocarpine, work by contraction of the ciliary muscle, opening the trabecular meshwork and allowing increased outflow of the aqueous humour. Echothiophate, an acetylcholinesterase inhibitor, is used in chronic glaucoma.
- Carbonic anhydrase inhibitors, such as dorzolamide, brinzolamide, and acetazolamide, lower secretion of aqueous humor by inhibiting carbonic anhydrase in the ciliary body.

1.10.7.2 Surgery

Both laser and conventional surgeries are performed to treat glaucoma. Surgery is the primary therapy for those withcongenital glaucoma. Generally, these operations are a temporary solution, as there is not yet a cure for glaucoma.

Canaloplasty

Canaloplasty is a nonpenetrating procedure using microcatheter technology. To perform a canaloplasty, an incision is made into the eye to gain access to the Schlemm's canal in a similar fashion to a viscocanalostomy. A microcatheter will circumnavigate the canal around the iris, enlarging the main drainage channel and its smaller collector channels through the injection of a sterile, gel-like material called viscoelastic. The catheter is then removed and a suture is placed within the canal and tightened.

By opening the canal, the pressure inside the eye may be relieved, although the reason is unclear, since the canal (of Schlemm) does not have any significant fluid resistance in glaucoma or healthy eyes. Long-term results are not available.

Laser surgery

Argon laser trabeculoplasty (ALT) may be used to treat open-angle glaucoma. It is a temporary solution, not a cure. A 50-µm argon laser spot is aimed at the trabecular meshwork to stimulate opening of the mesh to allow more outflow of aqueous fluid. Usually, half of the angle is treated at a time. Traditional laser trabeculoplasty uses a thermal argon laser in an argon laser trabeculoplasty procedure.

A newer type of laser trabeculoplasty uses a "cold" (nonthermal) laser to stimulate drainage in the trabecular meshwork. This newer procedure, selective laser trabeculoplasty (SLT), uses a 532-nm, frequency-doubled, Q-switched Nd:YAG laser, which selectively targets melanin pigment in the trabecular meshwork cells. Studies show SLT is as effective as

ALT at lowering eye pressure. In addition, SLT may be repeated three to four times, whereas ALT can usually be repeated only once.

Nd:YAG laser peripheral iridotomy (LPI) may be used in patients susceptible to or affected by angle closure glaucoma or pigment dispersion syndrome. During laser iridotomy, laser energy is used to make a small, full-thickness opening in the iris to equalize the pressure between the front and back of the iris, thus correcting any abnormal bulging of the iris. In people with narrow angles, this can uncover the trabecular meshwork. In some cases of intermittent or short-term angle closure, this may lower the eye pressure. Laser iridotomy reduces the risk of developing an attack of acute angle closure. In most cases, it also reduces the risk of developing chronic angle closure or of adhesions of the iris to the trabecular meshwork.

Diode laser cycloablation lowers IOP by reducing aqueous secretion by destroying secretory ciliary epithelium (Rosentreter *et al*., 2010).

Trabeculectomy

The most common conventional surgery performed for glaucoma is the trabeculectomy. Here, a partial thickness flap is made in the scleral wall of the eye, and a window opening is made under the flap to remove a portion of the trabecular meshwork. The scleral flap is then sutured loosely back in place to allow fluid to flow out of the eye through this opening, resulting in lowered intraocular pressure and the formation of a bleb or fluid bubble on the surface of the eye. Scarring can occur around or over the flap opening, causing it to become less effective or lose effectiveness altogether. Traditionally, chemotherapeutic adjuvants, such asmitomycin C (MMC, 0.5–0.2 mg/ml) or 5-fluorouracil (5-FU, 50 mg/ml), are applied with soaked sponges on the wound bed to prevent filtering blebs from scarring by inhibiting fibroblast proliferation. Contemporary alternatives include the sole or combinative implementation of nonchemotherapeutic adjuvants, such as collagen matrix implant or other biodegradable spacers, to prevent super scarring by randomization and modulation of fibroblast proliferation in addition to the mechanical prevention of wound contraction and adhesion (Akbari *et al*., 2009).

Glaucoma drainage implants

Professor Anthony Molteno developed the first glaucoma drainage implant, in Cape Town in 1966. Since then, several different types of implants have followed on from the original, the Baerveldt tube shunt, or the valved implants, such as the Ahmed glaucoma valve implant or the ExPress Mini Shunt and the later generation pressure ridge Molteno implants. These are indicated for glaucoma patients not responding to maximal medical therapy, with previous failed guarded filtering surgery (trabeculectomy). The flow tube is inserted into the anterior chamber of the eye, and the plate is implanted underneath the conjunctiva to allow flow of aqueous fluid out of the eye into a chamber called a bleb.

- The first-generation Molteno and other nonvalved implants sometimes require the ligation of the tube until the bleb formed is mildly fibrosed and water-tight. This is done to reduce postoperative hypotony—sudden drops in postoperative intraocular pressure.
- Valved implants, such as the Ahmed glaucoma valve, attempt to control postoperative hypotony by using a mechanical valve.
- Ab interno implants, such as the Xen Gel Stent, are transscleral implants by an ab interno procedure to channel aqueous humor into the non-dissected Tenon's space, creating a subconjunctival drainage area similar to a bleb. The implants are transscleral and different from more other ab interno implants that do not create a transscleral drainage, such as iStent, CyPass, or Hydrus (Roberts *et al*., 2010).

The ongoing scarring over the conjunctival dissipation segment of the shunt may become too thick for the aqueous humor to filter through. This may require preventive measures using antifibrotic medications, such as 5-fluorouracil or mitomycin-C (during the procedure), or other nonantifibrotic medication methods, such as collagen matrix implant, or biodegradable spacer, or later on create a necessity for revision surgery with the sole or combinative use of donor patch grafts or collagen matrix implant. And for glaucomatous painful blind eye and some cases of glaucoma, cyclocryotherapy for ciliary body ablation could be considered to be performed.

Veterinary implant

TR BioSurgical has commercialized a new implant specifically for veterinary medicine, called TR-ClarifEYE. The implant consists of a new biomaterial, the STAR BioMaterial, which consists of silicone with a very precise homogenous pore size, a property which reduces fibrosis and improves tissue integration. The implant contains no valves and is placed completely within the eye without sutures. To date, it has demonstrated long-term success (longer than a year) in a pilot study in medically refractory dogs with advanced glaucoma (Anisimova *et al*., 2012).

Laser-assisted nonpenetrating deep sclerectomy

The most common surgical approach currently used for the treatment of glaucoma is trabeculectomy, in which the sclera is punctured to alleviate intraocular pressure.

Nonpenetrating deep sclerectomy (NPDS) surgery is a similar, but modified, procedure, in which instead of puncturing the scleral bed and trabecular meshwork under a scleral flap, a second deep scleral flap is created, excised, with further procedures of deroofing the Schlemm's canal, upon which, percolation of liquid from the inner eye is achieved and thus alleviating intraocular pressure, without penetrating the eye. NPDS is demonstrated to cause significantly fewer side effects than trabeculectomy. However, NPDS is performed manually and requires higher level of skills that may be assisted with instruments. In order to prevent wound adhesion after deep scleral excision and to maintain good filtering results, NPDS as with other non-penetrating procedures is sometimes performed with a variety of biocompatible spacer or devices, such as the Aquaflow collagen wick, ologen Collagen Matrix, or Xenoplast glaucoma implant (Allen, 2006).

Laser-assisted NPDS is performed with the use of a CO_2 laser system. The laser-based system is self-terminating once the required scleral thickness and adequate drainage of the intraocular fluid have been achieved. This self-regulation effect is achieved as the CO_2 laser essentially stops ablating as soon as it comes in contact with the intraocular percolated liquid, which occurs as soon as the laser reaches the optimal residual intact layer thickness.

1.11 Cataract

A cataract is a clouding of the lens in the eye leading to a decrease in vision. It can affect one or both eyes. Often it develops slowly. Symptoms may include faded colors, blurry vision, and halos around light, trouble with bright lights, and trouble seeing at night. [33] This may result in trouble driving, reading, or recognizing faces. Poor vision may also result in an increased risk of falling and depression. Cataracts are the cause of half of blindness and 33% of visual impairment worldwide (Rao *et al*., 2011).

Cataracts are most commonly due to aging, but may also occur due to trauma, radiation exposure, be present from birth, or occur following eye surgery for other problems. Risk factors include diabetes, smoking tobacco, prolonged exposure to sunlight, and alcohol. Either clumps of protein or yellow-brown pigment may be deposited in the lens reducing the transmission of light to the retina at the back of the eye. Diagnosis is by an eye examination (WHO, 2012).

Prevention includes wearing sunglasses and not smoking. Early on the symptoms may be improved with eyeglasses. If this does not help, surgery to remove the cloudy lens and replace it with an artificial lens is the only effective treatment. Surgery is only needed if the cataracts are causing problems. Surgery generally results in an improved quality of life. Cataract surgery is not easily available in many countries, which is especially true of women.

About 20 million people globally are blind due to cataracts. It is the cause of about 5% of blindness in the United States and nearly 60% of blindness in parts of Africa and South America.[8] Blindness from cataracts occurs in about 10 to 40 per 100,000 children in the developing world and 1 to 4 per 100,000 children in the developed world. Cataracts become more common with age. About half the people in the United States have had cataracts by the age of 80.

1.11.1 Sign and symptoms

Signs and symptoms vary depending on the type of cataract, though considerable overlap occurs. People with nuclear sclerotic or brunescent cataracts often notice areduction of vision. Those with posterior subcapsular cataracts usually complain of glareas their major symptom.

The severity of cataract formation, assuming no other eye disease is present, is judged primarily by a visual acuity test. The appropriateness of surgery depends on a patient's particular functional and visual needs and other risk factors, all of which may vary widely (Duker *et al*., 2009).

Figure 1.3: normal and cataract vision (source: National eye institute, http://www.nei.nih.gov/photo/keyword.asp?narrow=Eye+Disease+Simulation&match=all)

1.11.2 Causes

Age

Age is the most common cause. Lens proteins denature and degrade over time, and this process is accelerated by diseases such as diabetes mellitus and hypertension. Environmental factors, including toxins, radiation, and ultraviolet light, have cumulative effects, which are worsened by the loss of protective and restorative mechanisms due to alterations in gene expression and chemical processes within the eye (Reddy, 1999).

Trauma

Blunt trauma causes swelling, thickening, and whitening of the lens fibers. While the swelling normally resolves with time, the white color may remain. In severe blunt trauma, or injuries which penetrate the eye, the capsule in which the lens sits can be damaged. This allows water from other parts of the eye to rapidly enter the lens leading to swelling and then whitening, obstructing light from reaching the retina at the back of the eye. Cataracts may develop in 0.7 to 8.0% of cases following electrical injuries (Sliney, 1994).

Radiation

Ultraviolet light, specifically UVB, has been shown to cause cataracts, and some evidence indicates sunglasses worn at an early age can slow its development in later life. The lens filters UV light; so once it is removed via surgery, one may be able to see UV light. Microwave radiation has also been found to cause cataracts. The mechanism is unclear, but may include changes in heat-sensitive enzymes that normally protect cell proteins in the lens. Another possible mechanism is direct damage to the lens from pressure waves induced in the aqueous humor. Cataracts have also been associated with ionizing radiation such as X-rays. [39] The addition of damage to the DNA of the lens cells also has been considered. Finally, electric and heat injuries denature and whiten the lens as a result of direct protein coagulation. This same process makes the clear albumin of an egg become white and opaque after cooking. Cataracts

of this type are often seen in glassblowers and furnace workers. Lasers of sufficient power output are known to damage the eyes and skin (Christen *et al*., 1992).

Genetics

The genetic component is strong in the development of cataracts, most commonly through mechanisms that protect and maintain the lens. The presence of cataracts in childhood or early life can occasionally be due to a particular syndrome. Examples of chromosome abnormalities associated with cataracts include 1q21.1 deletion syndrome, cri-du-chat syndrome, Down syndrome, Patau's syndrome, trisomy 18 (Edward's syndrome), and Turner's syndrome. Examples of single-gene disorderinclude Alport's syndrome, Conradi's syndrome, myotonic dystrophy, and oculocerebrorenal syndrome or Lowe syndrome.

Skin diseases

The skin and the lens have the same embryological origin and can be affected by similar diseases. Those with atopic dermatitis and eczema occasionally develop shield ulcers cataracts. Ichthyosis is an autosomal recessive disorder associated with cuneiform cataracts and nuclear sclerosis. Basal-cell nevus and pemphigus have similar associations.

Drug use

Cigarette smoking has been shown to double the rate of nuclear sclerotic cataracts and triple the rate of posterior sub capsular cataracts. (Brûle *et al*., 1998) Evidence is conflicting over the effect of alcohol. Some surveys have shown a link, but others which followed patients over longer terms have not.

Medications

Some drugs, such as corticosteroids, can induce cataract development. People with schizophrenia often have risk factors for lens opacities (such as diabetes, hypertension, and poor nutrition) but antipsychotic medications are unlikely to contribute to cataract formation. Miotics and triparanol may increase the risk (Bollinger *et al*., 2008).

Iatrogenic

Nearly every person who undergoes a vitrectomy - without ever having had cataract surgery - will experience progression of nuclear sclerosis at 6-months and 12-month after the operation. This may be because the native vitreous humour contains ascorbic acid which helps neutralize oxidative damage to the lens. Traditional vitreous substitutes such as balanced salt solution do not contain ascorbic acid. As such, for phakic patients requiring a vitrectomy it is becoming increasingly common for ophthalmologists to offer the vitrectomy with a combined prophylactic cataract surgery procedure to prophylactically prevent cataract formation (Greiner *et al*., 1979).

1.11.3 Classification

Cataracts may be partial or complete, stationary or progressive, or hard or soft. The main types of age-related cataracts are nuclear sclerosis, cortical, and posterior sub capsular. Nuclear sclerosis, the most common type of cataract, involves the central or 'nuclear' part of the lens. Over time, this becomes hard or 'sclerotic' due to condensation of lens nucleus and deposition of brown pigment within the lens. In advanced stages, it is called brunescent cataract. This type of cataract can present with a shift to nearsightedness and causes problems with distance vision, while reading is less affected (Yanoff *et al*., 2008).

Cortical cataracts are due to the lens cortex (outer layer) becoming opaque. They occur when changes in the water content of the periphery of the lens causes fissuring. When these cataracts are viewed through an ophthalmoscope or other magnification system, the appearance is similar to white spokes of a wheel. Symptoms often include problems with glare and light scatter at night. Posterior subcapsular cataracts are cloudy at back of the lens adjacent to the capsule (or bag) in which the lens sits. Because light becomes more focused toward the back of the lens, they can cause disproportionate symptoms for their size.

An immature cataract has some transparent protein, but with a mature cataract, all the lens protein is opaque. In a hypermature or Morgagnian cataract, the lens proteins have become liquid. Congenital cataract, which may be detected in adulthood, has a different classification

and includes lamellar, polar, and sutural cataracts. Cataracts can be classified by using the lens opacities classification system LOCS III. In this system, cataracts are classified based on type as nuclear, cortical, or posterior. The cataracts are further classified based on severity on a scale from 1 to 5. The LOCS III system is highly reproducible (Neale *et al*., 2009).

1.11.4 Prevention

Risk factors such as UVB exposure and smoking can be addressed, but are unlikely to make large difference to visual function. Although no means of preventing cataracts has been scientifically proven, wearing ultraviolet-protecting sunglasses may slow the development. While regular intake of antioxidants (such as vitamins A, C, and E) has been thought to protect against the risk of cataracts, clinical trials have shown it does not. Evidence is mixed, but weakly positive, for a potential protective effect of the nutrients lutein and zeaxanthin. Statin use is somewhat associated with a lower risk of nuclear sclerotic cataract (WHO, 2008).

1.11.5 Epidemiology

Age-related cataracts are responsible for 51% of world blindness, about 20 million people. Globally, cataracts cause moderate to severe disability in 53.8 million (2004), 52.2 million of whom are in low and middle income countries. In many countries, surgical services are inadequate, and cataracts remain the leading cause of blindness. Even where surgical services are available, low vision associated with cataracts may still be prevalent as a result of long waits for, and barriers to, surgery, such as cost, lack of information and transportation problems.

In the United States, age-related lens changes have been reported in 42% between the ages of 52 and 64, 60% between the ages 65 and 74, and 91% between the ages of 75 and 85. Cataracts affect nearly 22 million Americans age 40 and older. By age 80, more than half of all Americans have cataracts. Direct medical costs for cataract treatment are estimated at $6.8 billion annually. In the eastern Mediterranean region, cataracts are responsible for over 51% of blindness. Access to eye care in many countries in this region is limited (Black *et al*., 2008).

1.11.6 Treatment

Surgical

Cataract removal can be performed at any stage and no longer requires ripening of the lens. Surgery is usually 'outpatient' and performed using local anesthesia. About 9 of 10 patients can achieve a corrected vision of 20/40 or better after surgery.

Several recent evaluations found surgery can only meet expectations when significant functional impairment from poor vision exists prior to surgery. Visual function estimates such as VF-14 have been found to give more realistic estimates than visual acuity testing alone. In some developed countries, a trend to overuse cataract surgery has been noted, which may lead to disappointing results (Olsen *et al*., 2012).

Phacoemulsification is the most widely used cataract surgery. This procedure uses ultrasonic energy to emulsify the cataract lens. Phacoemulsification typically comprises six steps:

- Anaesthetic - The eye is numbed with either a subtenon injection around the eye or using simple eye drops.
- Corneal incision - Two cuts are made through the clear cornea to allow insertion of instruments into the eye.
- Capsulorhexis - A needle or small pair of forceps is used to create a circular hole in the capsule in which the lens sits.
- Phacoemulsification - A handheld probe is used to break up and emulsify the lens into liquid using the energy of ultrasound waves. The resulting 'emulsion' is sucked away.
- Irrigation and aspiration - The cortex, which is the soft outer layer of the cataract, is aspirated or sucked away. Fluid removed is continually replaced with a saline solution to prevent collapse of the structure of the anterior chamber (the front part of the eye).
- Lens insertion - A plastic, foldable lens is inserted into the capsular bag that formerly contained the natural lens. Some surgeons also inject an antibiotic into the eye to reduce the risk of infection. The final step is to inject salt water into the corneal wounds to cause the area to swell and seal the incision.

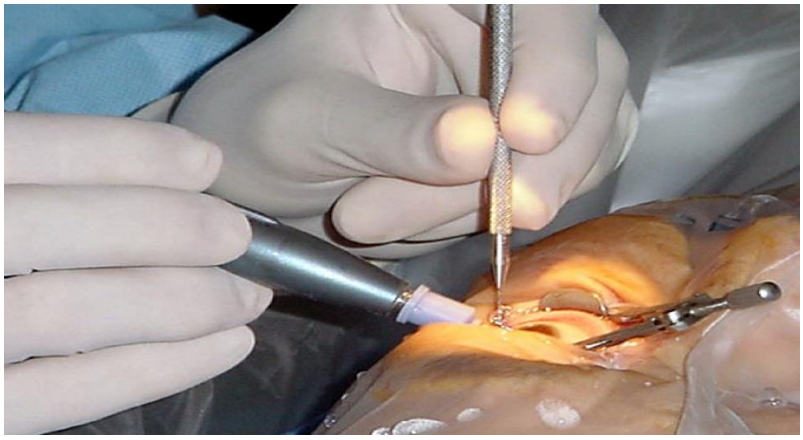


Figure 1.4: Cataract surgery (source: navy medical center, https://en.wikipedia.org/wiki/Cataract#/media/File:Cataract_surgery.jpg)

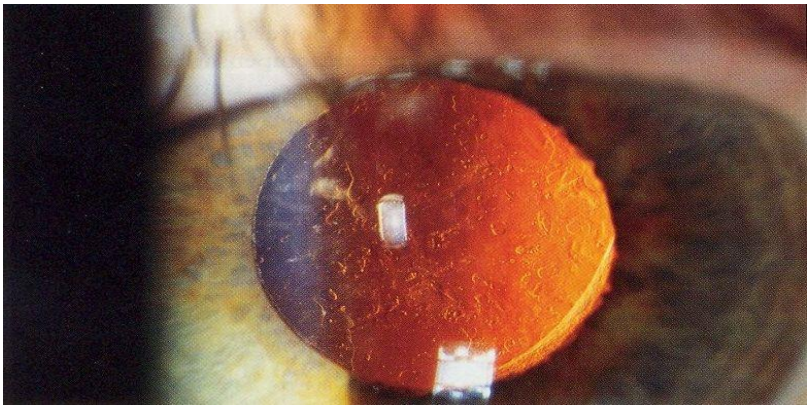


Figure 1.5: posterior capsular opacification (source: https://en.wikipedia.org/wiki/Cataract#/media/File:Posterior_capsular_opacification_on_retroillumination.jpg)

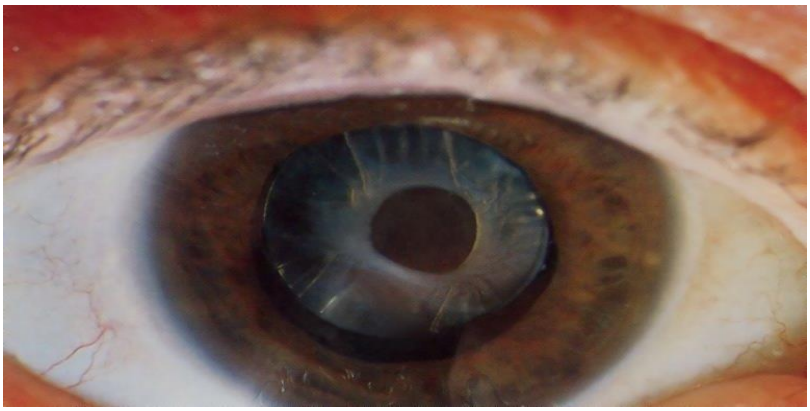


Figure 1.6: anterior capsular opacification (source: https://en.wikipedia.org/wiki/Cataract#/media/File:Anterior_capsular_opacification.jpg)

Extra capsular cataract extraction (ECCE) consists of removing the lens manually, but leaving the majority of the capsule intact. The lens is expressed through a 10– to 12-mm incision which is closed with sutures at the end of surgery. ECCE is less frequently performed than phacoemulsification, but can be useful when dealing with very hard cataracts or other situations where emulsification is problematic. Manual small incision cataract surgery (MSICS) has evolved from ECCE. In MSICS, the lens is removed through a self-sealing scleral tunnel wound in the sclera which, ideally, is watertight and does not require suturing. Although "small", the incision is still markedly larger than the portal in phacoemulsion. This surgery is increasingly popular in the developing world where access to phacoemulsification is still limited.

Intracapsular cataract extraction (ICCE) is rarely performed. The lens and surrounding capsule are removed in one piece through a large incision while pressure is applied to the vitreous membrane. The surgery has a high rate of complications.

1.11.7 Prognosis

Postoperative care

The postoperative recovery period (after removing the cataract) is usually short. The patient is usually ambulatory on the day of surgery, but is advised to move cautiously and avoid straining or heavy lifting for about a month. The eye is usually patched on the day of surgery and use of an eye shield at night is often suggested for several days after surgery.

In all types of surgery, the cataractous lens is removed and replaced with an artificial lens, known as an intraocular lens, which stays in the eye permanently. Intraocular lenses are usually monofocal, correcting for either distance or near vision. Multifocal lenses may be implanted to improve near and distance vision simultaneously, but these lenses may increase the chance of unsatisfactory vision.

Complications

Serious complications of cataract surgery are retinal detachment and endophthalmitis. In both cases, patients notice a sudden decrease in vision. In endophthalmitis, patients often describe pain. Retinal detachment frequently presents with unilateral visual field defects, blurring of vision, flashes of light, or floating spots.

The risk of retinal detachment was estimated at about 0.4% within 5.5 years, corresponding to a 2.3-fold risk increase compared to naturally expected incidence, with older studies reporting a substantially higher risk. The incidence is increasing over time in a somewhat linear manner, and the risk increase lasts for at least 20 years after the procedure. Particular risk factors are younger age, male sex, longer axial length, and complications during surgery. In the highest risk group of patients, the incidence of pseudophakic retinal detachment may be as high as 20%. The risk of endophthalmitis occurring after surgery is less than one in 1000 (Williams *et al*., 2006).

Corneal oedema and cystoid macular oedema are less serious but more common, and occur because of persistent swelling at the front of the eye in corneal oedema or back of the eye in cystoid macular oedema. They are normally the result of excessive inflammation following surgery, and in both cases, patients may notice blurred, foggy vision. They normally improve with time and with application of anti-inflammatory drops. The risk of either occurring is around one in 100 (RNIB, 2012).

Posterior capsular opacification, also known as after-cataract, is a condition in which months or years after successful cataract surgery, vision deteriorates or problems with glare and light scattering recur, usually due to thickening of the back or posterior capsule surrounding the implanted lens, so-called 'posterior lens capsule opacification'. Growth of natural lens cells remaining after the natural lens was removed may be the cause, and the younger the patient, the greater the chance of this occurring. Management involves cutting a small, circular area in the posterior capsule with targeted beams of energy from a laser, called Nd:YAG lasercapsulotomy, after the type of laser used. The laser can be aimed very accurately, and the small part of the capsule which is cut falls harmlessly to the bottom of the inside of the eye. This procedure leaves sufficient capsule to hold the lens in place, but removes enough to allow light to pass directly through to the retina. Serious side effects are rare. Posterior capsular opacification is common and occurs following up to one in four operations, but these rates are decreasing following the introduction of modern intraocular lenses together with a better understanding of the causes. Vitreous touch syndrome is a possible complication of intracapsular cataract extraction (The Economist, 2015).

1.12 Macular degeneration

Macular degeneration, often age-related macular degeneration (AMD or ARMD), is a medical condition that usually affects older adults and results in a loss of vision in the center of the visual field (the macula) because of damage to the retina. It occurs in "dry" and "wet" forms. It is a major cause of blindness and visual impairment in older adults, afflicting 30-50 million people globally. Macular degeneration can make it difficult or impossible to read or recognize faces, although enough peripheral vision remains to allow other activities of daily life (PT, 2006).

Although some macular dystrophies affecting younger individuals are sometimes rarely referred to as macular degeneration, the term generally refers to age-related macular degeneration (AMD or ARMD). The retina is a network of visual receptors and nerves. It lies on the choroid, a network of blood vessels that supply the retina with blood.

In the dry (no exudative) form, cellular debris called drusen accumulates between the retina and the choroid, causing atrophy and scarring to the retina. In the wet (exudative) form, which is more severe, blood vessels grow up from the choroid behind the retina which can leak exudate and fluid and also cause hemorrhaging. It can be treated with laser coagulation, and more commonly with medication that stops and sometimes reverses the growth of blood vessels (Roberts, 2006).

1.12.1 Sign and symptoms

Signs and symptoms of macular degeneration include:

- Drusen, tiny accumulations of extracellular material that build up on the retina
- Pigmentary changes
- Distorted vision in the form of metamorphopsia, in which a grid of straight lines appears wavy and parts of the grid may appear blank: Patients often first notice this when looking at things like miniblinds in their home or telephone poles while driving.

- Exudative changes: hemorrhages in the eye, hard exudates, subretinal/sub-RPE/intraretinal fluid
- Slow recovery of visual function after exposure to bright light (photostress test)
- Atrophy: incipient and geographic
- Visual acuity drastically decreasing (two levels or more), e.g.: 20/20 to 20/80
- Preferential hyperacuity perimetry changes (for wet AMD)

- Blurred vision: Those with nonexudative macular degeneration may be asymptomatic or notice a gradual loss of central vision, whereas those with exudative macular degeneration often notice a rapid onset of vision loss (often caused by leakage and bleeding of abnormal blood vessels).
- Central scotomas (shadows or missing areas of vision)
- Trouble discerning colors, specifically dark ones from dark ones and light ones from light ones
- A loss in contrast sensitivity
- Straight lines appear curved in an Amsler grid.

Macular degeneration by itself will not lead to total blindness. For that matter, only a very small number of people with visual impairment are totally blind. In almost all cases, some vision remains, mainly peripheral. Other complicating conditions may possibly lead to such an acute condition (severe stroke or trauma, untreated glaucoma, etc.), but few macular degeneration patients experience total visual loss.

The area of the macula comprises only about 2.1% of the retina, and the remaining 97.9% (the peripheral field) remains unaffected by the disease. Interestingly, even though the macula provides such a small fraction of the visual field, almost half of the visual cortex is devoted to processing macular information (Allday, 2012).

The loss of central vision profoundly affects visual functioning. It is quite difficult, for example, to read without central vision. Pictures that attempt to depict the central visual loss of macular

degeneration with a black spot do not really do justice to the devastating nature of the visual loss. This can be demonstrated by printing letters six inches high on a piece of paper and attempting to identify them while looking straight ahead and holding the paper slightly to the side. Most people find this difficult to do.

There is a loss of contrast sensitivity, so that contours, shadows, and color vision are less vivid. The loss in contrast sensitivity can be quickly and easily measured by a contrast sensitivity test performed either at home or by an eye specialist.

Similar symptoms with a very different etiology and different treatment can be caused by epiretinal membrane or macular pucker or any other condition affecting the macula, such as central serous retinopathy.

Like many age-related disorders, macular degeneration was once rare, but has become increasingly common in developed nations in the last 30 years due to the rapid growth in the number of people over 75 and poses a significant health care challenge as it is the most common cause of vision loss in adults, with the bulk of new cases occurring after the age of 80. By 2020, it is estimated that as many as 3 million Americans will be affected (Hirschler *et al*., 2008).

1.12.2 Risk factor

Aging: About 10% of patients 66 to 74 years of age will have findings of macular degeneration. The prevalence increases to 30% in patients 75 to 85 years of age.

Family history: The lifetime risk of developing late-stage macular degeneration is 50% for people who have a relative with macular degeneration, versus 12% for people who do not have relatives with macular degeneration. Researchers from the University of Southampton reported they had discovered six mutations of the gene SERPING1that are associated with AMD. Mutations in this gene can also cause hereditary angioedema (Dasari *et al*., 2011).

Hypertension (high blood pressure) Individuals with high blood pressure are more likely to be affected by age-related macular degeneration. This is because high blood pressure, like smoking, leads to a constriction, or narrowing, of the blood vessels in the choroid which gives blood and oxygen to the retina. This constriction limits the amount of blood able to circulate and give blood and oxygen to the retina and therefore having a negative effect on its health.

Cholesterol: Elevated cholesterol may increase the risk of AMD (Chew *et al*., 2007).

Obesity: Abdominal obesity is a risk factor, especially among men.

Fat intake consuming high amounts of certain fats likely contributes to AMD, while monounsaturated fats are potentially protective. In particular, ω-3 fatty acids may decrease the risk of AMD (Roberts, 2005).

Oxidative stress: Age-related accumulation of low-molecular-weight, phototoxic, pro-oxidant melanin oligomers within lysosomes in the retinal pigment epithelium may be partly responsible for decreasing the digestive rate of photoreceptor outer rod segments (POS) by the RPE. A decrease in the digestive rate of POS has been shown to be associated with lipofuscin formation - a classic sign associated with AMD.

Fibulin-5 mutation: Rare forms of the disease are caused by genetic defects in fibulin-5, in an autosomal dominant manner. In 2004, Stone *et al*. performed a screen on 402 AMD patients and revealed a statistically significant correlation between mutations in fibulin-5 and incidence of the disease. Furthermore, the point mutants were found in the calcium-binding sites of the cbEGF domains of the protein. There is no structural basis for the effects of the mutations.

Race: Macular degeneration is more likely to be found in Caucasians than in people of African descent.

Exposure to sunlight, especially blue light: Evidence is conflicting as to whether exposure to sunlight contributes to the development of macular degeneration. A recent study on 446

subjects found it does not. Other research, however, has shown high-energy visible light may contribute to AMD.

Vitamin D deficiency: higher vitamin D levels are associated with lower age-related macular degeneration risk in women.

Smoking: Smoking tobacco increases the risk of AMD by two to three times that of someone who has never smoked, and may be the most important modifiable factor in its prevention. A review of previous studies found "the literature review confirmed a strong association between current smoking and AMD. ... Cigarette smoking is likely to have toxic effects on the retina (Best *et al*., 2011).

1.12.3 Pathophysiology

Starting from the inside of the eye and going towards the outer surface, the three main layers at the back of the eye are theretina, which is light-sensitive tissue that is considered part of the central nervous system and is actually brain tissue; thechoroid, which is made up of a web of blood vessels; and the sclera, which is the tough, white, outer layer of the eye.

Dry AMD

Age-related macular degeneration begins with characteristic yellow deposits (drusen) in the macula, between the retinal pigment epithelium and the underlying choroid. Most people with these early changes (referred to as age-related maculopathy) still have good vision. People with drusen may or may not develop AMD, in fact the majority of people over age 55 have drusen with no negative effects. The risk of developing symptoms is higher when the drusen are large and numerous and associated with disturbance in the pigmented cell layer under the macula. Large and soft drusen are thought to be related to elevated cholesterol deposits.

Central geographic atrophy, the "dry" form of advanced AMD, results from atrophy of the retinal pigment epithelial layer below the retina, which causes vision loss through loss of photoreceptors (rods and cones) in the central part of the eye.

Wet AMD

Neovascular or exudative AMD, the "wet" form of advanced AMD, causes vision loss due to abnormal blood vessel growth (choroidal neovascularization) in thechoriocapillaris, through Bruch's membrane. The proliferation of abnormal blood vessels in the retina is stimulated by vascular endothelial growth factor (VEGF). Unfortunately, these new vessels are fragile, ultimately leading to blood and protein leakage below the macula. Bleeding, leaking, and scarring from these blood vessels eventually because irreversible damage to the photoreceptors and rapid vision loss if left untreated. Only about 10% of patients suffering from macular degeneration have the wet type.

1.12.4 Diagnosis

The major symptoms of macular degeneration:

- When viewing an Amsler grid, some straight lines appear wavy and some patches appear blank
- When viewing a Snellen chart, at least 2 lines decline
- In dry macular degeneration, which occurs in 85-90 percent of AMD cases, drusen spots can be seen in Fundus photography
- In wet macular degeneration, using angiography we can see leakage of bloodstream behind the macula
- Using an electroretinogram, points in the macula with a weak or absent response compared to a normal eye may be found
- Visual acuity and color sensitivity should be similar for red, green and blue (RGB)

Fluorescein angiography allows for the identification and localization of abnormal vascular processes. Optical coherence tomography is now used by most ophthalmologists in the diagnosis and the follow-up evaluation of the response to treatment by using either bevacizumab (Avastin) or ranibizumab (Lucentis), which are injected into the vitreous humor of the eye at various intervals.

Recently, structured illumination light microscopy using a specially designed super resolution microscope setup has been used to resolve the fluorescent distribution of small autofluorescent structures (lipofuscin granulae) in retinal pigment epithelium tissue sections (Tan *et al*., 2008).

1.12.5 Management

Dry AMD

No medical or surgical treatment is available for this condition; however, the AREDS trial found benefits with some vitamin supplements along with high doses of antioxidants. The follow up study, AREDS2, showed that the antioxidants lutein and zeaxanthin also have benefits. These combinations of supplements have been suggested by the National Eye Institute and others to slow progression of the disease in people who have intermediate AMD, and those who have late AMD in one eye. Though, the researchers stress that the AREDS formulation is not a cure, and will not restore vision already lost from AMD. The studies didn't prove that it helps people with early AMD, but it is reasonable to suggest that the benefits of the supplements also extend to those with early AMD. But, not all antioxidants are beneficial, higher beta-carotene intake was associated with an increased risk of AMD in addition to its association with increased lung cancers in smokers.

Wet AMD

Due to the involvement by vascular endothelial growth factor (VEGF) in the development of new blood vessels, antiangiogenics or anti-VEGF agents can cause regression of the abnormal blood vessels and improve vision when injected directly into the vitreous humor of the eye. The injections must be repeated monthly or bimonthly initially, but the treatment may be stopped if

the condition becomes stabilized. Several antiangiogenic drugs have been approved for use in the eye by theFDA and regulatory agencies in other countries.

The first angiogenesis inhibitor, a monoclonal antibody against VEGF-A, was bevacizumab, which is approved for use in several cancers. Ranibizumab is a smaller fragment, Fab fragment, of the parent bevacizumab molecule specifically designed for eye injections. A controversy in the UK involved the off-label use of cheaper bevacizumab over the approved, but expensive, ranibizumab. A recent randomized control trial found that bevacizumab and ranibizumab had similar efficacy, and reported no significant increase in adverse events with bevacizumab. A 2014 Cochrane review found that the systemic safety of bevacizumab and ranibizumab are similar when used to treat neovascular AMD, except for gastrointestinal disorders. Other approved antiangiogenic drugs for the treatment of neo-vascular AMD include pegaptanib and aflibercept. Photodynamic therapy has also been used to treat wet AMD. The drug verteporfin is administered intravenously; light of a certain wavelength is then applied to the abnormal blood vessels. This activates the verteporfin destroying the vessels (Evans *et al*., 2006).

Medications

A 2015 Cochrane review did not find enough evidence for statins.

Vitamins

A 2012 Cochrane review found the use of vitamin and mineral supplements, alone or in combination, by the general population had no effect on whether or not AMD started,[63] a finding echoed by another review. It; however, may slow down the worsening of AMD.[65] The review also questioned the possible harm of such supplements, given the increased risk of lung cancer in smokers with high intakes of beta-carotene, and the increased risk of heart failure in at-risk populations who consume high levels of vitamin E supplements (John, 2013).

Adaptive devices

Because peripheral vision is not affected, people with macular degeneration can learn to use their remaining vision to partially compensate. Assistance and resources are available in many

countries and every state in the U.S. Classes for "independent living" are given and some technology can be obtained from a state department of rehabilitation.

Adaptive devices can help people read. These include magnifying glasses, special eyeglass lenses, computer screen readers, and TV systems that enlarge reading material.

Video cameras can be fed into standard or special-purpose computer monitors, and the image can be zoomed in and magnified. These systems often include a movable table to move the written material. Accessible publishing provides larger fonts for printed books, patterns to make tracking easier, audiobooks and DAISY books with both text and audio (Kertes *et al*., 2007).

1.13 Diabetic retinopathy

Diabetic retinopathy also known as diabetic eye disease is when damage occurs to the retina due to diabetes. It can eventually lead to blindness. It is an ocular manifestation of diabetes, a systemic disease, which affects up to 80 percent of all patients who have had diabetes for 10 years or more. Despite these intimidating statistics, research indicates that at least 90% of these new cases could be reduced if there were proper and vigilant treatment and monitoring of the eyes. The longer a person has diabetes, the higher his or her chances of developing diabetic retinopathy. Each year in the United States, diabetic retinopathy accounts for 12% of all new cases of blindness. It is also the leading cause of blindness for people aged 20 to 64 years (Geiss *et al*., 2004).

1.13.1 Sign and symptoms

Diabetic retinopathy often has no early warning signs. Even macular edema, which can cause rapid vision loss, may not have any warning signs for some time. In general, however, a person with macular edema is likely to have blurred vision, making it hard to do things like read or drive. In some cases, the vision will get better or worse during the day.

In the first stage which is called non-proliferative diabetic retinopathy (NPDR) there are no symptoms, the signs are not visible to the eye and patients will have 20/20 vision. The only way to detect NPDR is by fundus photography, in which microaneurysms (microscopic blood-filled

bulges in the artery walls) can be seen. If there is reduced vision, fluoresceinangiography can be done to see the back of the eye. Narrowing or blocked retinal blood vessels can be seen clearly and this is called retinal ischemia (lack of blood flow).

Macular edema in which blood vessels leak their contents into the macular region can occur at any stage of NPDR. The symptoms of macular edema are blurred vision and darkened or distorted images that are not the same in both eyes. Ten percent (10%) of diabetic patients will have vision loss related to macular edema. Optical Coherence Tomography can show the areas of retinal thickening (due to fluid accumulation) of macular edema.

In the second stage, abnormal new blood vessels (neovascularisation) form at the back of the eye as part of proliferative diabetic retinopathy (PDR); these can burst and bleed (vitreous hemorrhage) and blur the vision, because these new blood vessels are fragile. The first time this bleeding occurs, it may not be very severe. In most cases, it will leave just a few specks of blood, or spots floating in a person's visual field, though the spots often go away after a few hours.These spots are often followed within a few days or weeks by a much greater leakage of blood, which blurs the vision. In extreme cases, a person may only be able to tell light from dark in that eye. It may take the blood anywhere from a few days to months or even years to clear from the inside of the eye, and in some cases the blood will not clear. These types of large hemorrhages tend to happen more than once, often during sleep. On funduscopic exam, a doctor will see cotton wool spots, flame hemorrhages (similar lesions are also caused by the alpha-toxin of Clostridium novyi), and dot-blot hemorrhages.

Figure 1.7: Normal vision (source: National eye institute, http://www.nei.nih.gov/photo/keyword.asp?narrow=Eye+Disease+Simulation&match=all)

Figure 1.8: The same view with diabetic retinopathy (source: National eye institute, http://www.nei.nih.gov/photo/keyword.asp?narrow=Eye+Disease+Simulation&match=all)

1.13.2 Risk factor

All people with diabetes mellitus are at risk – those with Type I diabetes and those with Type II diabetes. The longer a person has diabetes, the higher the risk of developing some ocular problem. Between 40 to 45 percent of Americans diagnosed with diabetes have some stage of diabetic retinopathy. After 20 years of diabetes, nearly all patients with Type I diabetes and >60% of patients with Type II diabetes have some degree of retinopathy; however, these statistics were published in 2002 using data from four years earlier, limiting the usefulness of the research. The subjects would have been diagnosed with diabetes in the late 1970s, before modern fast acting insulin and home glucose testing. Prior studies had also assumed a clear glycemic threshold between people at high and low risk of diabetic retinopathy (James *et al*., 1997).

However, it has been shown that the widely accepted WHO and American Diabetes Association diagnostic cutoff for diabetes of a fasting plasma glucose ≥ 7.0 mmol/l (126 mg/dl) does not accurately identify diabetic retinopathy among patients. The cohort study included a multi-ethnic, cross-sectional adult population sample in the US, as well as two cross-sectional adult populations in Australia. For the US-based component of the study, the sensitivity was 34.7% and specificitywas 86.6%. For patients at similar risk to those in this study (15.8% had diabetic retinopathy), this leads to a positive predictive value of 32.7% and negative predictive value of

87.6%. Published rates vary between trials, the proposed explanation being differences in study methods and reporting of prevalence rather than incidence values (Ryeom *et al*., 2009).

During pregnancy, diabetic retinopathy may also be a problem for women with diabetes. It is recommended that all pregnant women with diabetes have dilated eye examinations each trimester to protect their vision. People with Down's syndrome, who have three copies of chromosome 21, almost never acquire diabetic retinopathy. This protection appears to be due to the elevated levels of endostatin, an anti-angiogenic protein, derived from collagen XVIII. The collagen XVIII gene is located on chromosome 21 (Pardianto, 2005).

1.13.3 Pathogenesis

Diabetic retinopathy is the result of microvascular retinal changes. Hyperglycemia-induced intramural pericyte death and thickening of the basement membrane lead to incompetence of the vascular walls. These damages change the formation of the blood-retinal barrier and also make the retinal blood vessels become more permeable (Toke *et al*., 2010).

The pericyte death is caused when "hyperglycemia persistently activates protein kinase C-δ (PKC-δ, encoded by Prkcd) and p38 mitogen-activated protein kinase (MAPK) to increase the expression of a previously unknown target of PKC-δ signaling, Src homology-2 domain–containing phosphatase-1 (SHP-1), a protein tyrosine phosphatase. This signaling cascade leads toPDGF receptor- dephosphorylation and a reduction in downstream signaling from this receptor, resulting in pericyte apoptosis.

Small blood vessels – such as those in the eye – are especially vulnerable to poor blood sugar (blood glucose) control. An overaccumulation of glucose and/or fructose damages the tiny blood vessels in the retina. During the initial stage, called nonproliferative diabetic retinopathy (NPDR), most people do not notice any change in their vision. Early changes that are reversible and do not threaten central vision are sometimes termed simplex retinopathy or background retinopathy (NHS England, 2009).

Some people develop a condition called macular edema. It occurs when the damaged blood vessels leak fluid and lipids onto the macula, the part of the retina that lets us see detail. The fluid makes the macula swell, which blurs vision.

1.13.4 Diagnosis

Diabetic retinopathy is detected during an eye examination that includes:

- Visual acuity test: This test uses an eye chart to measure how well a person sees at various distances (i.e., visual acuity).
- Pupil dilation: The eye care professional places drops into the eye to dilate the pupil. This allows him or her to see more of the retina and look for signs of diabetic retinopathy. After the examination, close-up vision may remain blurred for several hours.
- Ophthalmoscopy or fundus photography: Ophthalmoscopy is an examination of the retina in which the eye care professional: (1) looks through a slit lamp biomicroscope with a special magnifying lens that provides a narrow view of the retina, or (2) wearing a headset (indirect ophthalmoscope) with a bright light, looks through a special magnifying glass and gains a wide view of the retina. Hand-held ophthalmoscopy is insufficient to rule out significant and treatable diabetic retinopathy. Fundus photography generally recreate considerably larger areas of the fundus, and has the advantage of photo documentation for future reference, as well as availing the image to be examined by a specialist at another location and/or time.
- Fundus Fluorescein angiography (FFA): This is an imaging technique which relies on the circulation of Fluorescein dye to show staining, leakage, or non-perfusion of the retinal and choroidal vasculature.
- Optical coherence tomography (OCT): This is an optical imaging modality based upon interference, and analogous to ultrasound. It produces cross-sectional images of the retina (B-scans) which can be used to measure the thickness of the retina and to resolve its major layers, allowing the observation of swelling.
- Digital Retinal Screening Programs: Systematic programs for the early detection of eye disease including diabetic retinopathy are becoming more common, such as in the UK,

where all people with diabetes are offered retinal screening at least annually. This involves digital image capture and transmission of the images to a digital reading center for evaluation and treatment referral. See Vanderbilt Ophthalmic Imaging Center and the NHS Diabetic Eye Screening Programme The name Diabetic Retinopathy Screening Service (DRSS) is also used (IANS, 2012).

- Computer Vision Approach: It is a System developed by Researchers at IIT Kharagpur in collaboration with IBM India. It uses data analytics capabilities to automatically compare and analyse retina images of the patient. It can tell if the patient has DR and also provides risk categorisation ranging from low to medium and high (Masharani *et al*., 2006).
- Slit Lamp Biomicroscopy Retinal Screening Programs: Systematic programs for the early detection of diabetic retinopathy using slit-lamp biomicroscopy. These exist either as a standalone scheme or as part of the Digital program (above) where the digital photograph was considered to lack enough clarity for detection and/or diagnosis of any retinal abnormality.

The eye care professional will look at the retina for early signs of the disease, such as:

1. leaking blood vessels,
2. retinal swelling, such as macular edema,
3. pale, fatty deposits on the retina (exudates) – signs of leaking blood vessels,
4. damaged nerve tissue (neuropathy), and
5. Any changes in the blood vessels.

If macular edema is suspected, FFA and sometimes OCT may be performed.

According to a DRSS user manual, poor quality images (which may apply to other methods) may be caused by cataract, poor dilation, ptosis, external ocular condition, or learning difficulties. There may be artefacts caused by dust, dirt, condensation, or smudge.

1.13.5 Management

There are three major treatments for diabetic retinopathy, which are very effective in reducing vision loss from this disease. In fact, even people with advanced retinopathy have a 90 percent chance of keeping their vision when they get treatment before the retina is severely damaged. These three treatments are laser surgery, injection of corticosteroids or anti-VEGF agents into the eye, and vitrectomy.

Although these treatments are very successful (in slowing or stopping further vision loss), they do not cure diabetic retinopathy. Caution should be exercised in treatment with laser surgery since it causes a loss of retinal tissue. It is often more prudent to inject triamcinolone or anti-VEGF drugs. In some patients it results in a marked increase of vision, especially if there is an edema of the macula. Avoiding tobacco use and correction of associated hypertension are important therapeutic measures in the management of diabetic retinopathy (O'Malley *et al*., 2012).

The best way of addressing diabetic retinopathy is to monitor it vigilantly and achieve euglycemia. Since 2008 there have been other therapies (e.g. kinase inhibitors and anti-VEGF) drugsavailable.

Laser photocoagulation

Laser photocoagulation can be used in two scenarios for the treatment of diabetic retinopathy. It can be used to treat macular edema by creating a Modified Grid at the posterior pole and it can be used for pan retinal coagulation for controlling neovascularization. It is widely used for early stages of proliferative retinopathy.

Modified Grid Laser photocoagulation

A 'C' shaped area around the macula is treated with low intensity small burns. This helps in clearing the macular edema.

Panretinal photocoagulation

Panretinal photocoagulation, or PRP (also called scatter laser treatment), is used to treat proliferative diabetic retinopathy (PDR). The goal is to create 1,600 - 2,000 burns in the retina with the hope of reducing the retina's oxygen demand, and hence the possibility of ischemia. It is done in multiple sittings. In treating advanced diabetic retinopathy, the burns are used to destroy the abnormal blood vessels that form in the retina. This has been shown to reduce the risk of severe vision loss for eyes at risk by 50%.

Before using the laser, the ophthalmologist dilates the pupil and applies anaesthetic drops to numb the eye. In some cases, the doctor also may numb the area behind the eye to reduce discomfort. The patient sits facing the laser machine while the doctor holds a special lens on the eye. The physician can use a single spot laser or a pattern scan laser for two dimensional patterns such as squares, rings and arcs. During the procedure, the patient will see flashes of light. These flashes often create an uncomfortable stinging sensation for the patient. After the laser treatment, patients should be advised not to drive for a few hours while the pupils are still dilated. Vision will most likely remain blurry for the rest of the day. Though there should not be much pain in the eye itself, an ice-cream headache like pain may last for hours afterwards.

Patients will lose some of their peripheral vision after this surgery although it may be barely noticeable by the patient. The procedure does however save the center of the patient's sight. Laser surgery may also slightly reduce colour and night vision. A person with proliferative retinopathy will always be at risk for new bleeding, as well as glaucoma, a complication from the new blood vessels. This means that multiple treatments may be required to protect vision.

Intravitreal triamcinolone acetonide

Triamcinolone is a long acting steroid preparation. When injected in the vitreous cavity, it decreases the macular edema (thickening of the retina at the macula) caused due to diabetic maculopathy, and results in an increase in visual acuity. The effect of triamcinolone is transient, lasting up to three months, which necessitates repeated injections for maintaining the beneficial effect. Best results of intravitreal Triamcinolone have been found in eyes that have

already undergonecataract surgery. Complications of intravitreal injection of triamcinolone include cataract, steroid-induced glaucoma and endophthalmitis.

Intravitreal anti-VEGF drugs

There are good results from multiple doses of intravitreal injections of anti-VEGF drugs such as bevacizumab. Present recommended treatment for diabetic macular edema is Modified Grid laser photocoagulation combined with multiple injections of anti-VEGF drugs (Quigley *et al*., 2006).

Vitrectomy

Instead of laser surgery, some people require a vitrectomy to restore vision. A vitrectomy is performed when there is a lot of blood in the vitreous. It involves removing the cloudy vitreous and replacing it with a saline solution.

Studies show that people who have a vitrectomy soon after a large hemorrhage are more likely to protect their vision than someone who waits to have the operation. Early vitrectomy is especially effective in people with insulin-dependent diabetes, who may be at greater risk of blindness from a hemorrhage into the eye.

Vitrectomy is often done under local anesthesia. The doctor makes a tiny incision in the sclera, or white of the eye. Next, a small instrument is placed into the eye to remove the vitreous and insert the saline solution into the eye.

Patients may be able to return home soon after the vitrectomy, or may be asked to stay in the hospital overnight. After the operation, the eye will be red and sensitive, and patients usually need to wear an eyepatch for a few days or weeks to protect the eye. Medicated eye drops are also prescribed to protect againstinfection. Vitrectomy is frequently combined with other modalities of treatment.

1.14 Global epidemiology

As of 2010, there were 44.7 million people in the world with open angle glaucoma. The same year, there were 2.8 million people in the United States with open angle glaucoma. By 2020,

the prevalence is projected to increase to 58.6 million worldwide and 3.4 million the United States (Akbari *et al*., 2009).

Internationally, glaucoma is the second-leading cause of blindness, after cataracts. Glaucoma is also the leading cause of blindness in African Americans, who have higher rates of primary open angle glaucoma. Bilateral vision loss can negatively affect mobility and interfere with driving. A meta-analysis published in 2009 found that people with primary open angle glaucoma do *not* have increased mortality rates, or increased risk of cardiovascular death (WHO, 2008).

Age-related cataracts are responsible for 51% of world blindness, about 20 million people. Globally, cataracts cause moderate to severe disability in 53.8 million (2004), 52.2 million of whom are in low and middle income countries (Sperduto *et al*., 1980).
In many countries, surgical services are inadequate, and cataracts remain the leading cause of blindness. Even where surgical services are available, low vision associated with cataracts may still be prevalent as a result of long waits for, and barriers to, surgery, such as cost, lack of information and transportation problems (Toh *et al*., 2007).

In the United States, age-related lens changes have been reported in 42% between the ages of 52 and 64, 60% between the ages 65 and 74, and 91% between the ages of 75 and 85. Cataracts affect nearly 22 million Americans age 40 and older. By age 80, more than half of all Americans have cataracts. Direct medical costs for cataract treatment are estimated at $6.8 billion annually.

In the eastern Mediterranean region, cataracts are responsible for over 51% of blindness. Access to eye care in many countries in this region is limited.

Chapter two

Literature review

2.1 Parental knowledge, attitude and practice related to blindness of children in some selected Upazilla of Bangladesh (Muhit *et al*., 2011).

Early detection of blind children at the household and community level is critical in reducing the global burden of visual impairment and childhood blindness. The aim of the study is to identify a range of potential issues relating to parental awareness and perceptions of common eye diseases affecting children. It was a descriptive and cross sectional study. Parents were recruited from four selected Upazillas ('pouroshoba' - 25% and rural - 75%) in the Naogaon district of Bangladesh. The method used in this study to assess parental knowledge and belief was by means of a questionnaire. The selected subjects were interviewed in detail using a structured questionnaire. It is mentionable that among common eye disease, about three-fourth of the parents informed that vitamin-A deficiency was the leading cause of blindness and more than one quarter believed thateye infection was the important cause of childhood blindness. Very few reported that injury in eye was the cause of childhood blindness. Analysis of respondents of this study revealed that half of the parents believed that childhood cataract is untreatable. Approximately 90% those surveyed were unaware of schooling systems for blind children and only 5% sought treatment from an ophthalmologist. This study also demonstrates that the health seeking behavior of parents and their extended families is poor. The mean age of the parents was 32.5±9.3 years, about 75% of parents had education up to primary level, and only 3.7% of them had graduation degree and above. The findings reinforce the necessity of parental awareness of common eye diseases in children and the importance of seeking timely advice including treatment based on informed decisions.

2.2 Awareness of eye diseases in an urban population in southern India (Rajesh *et al*., 2001).

Objective of this study was to assess the level of awareness of eye diseases in the urban population of Hyderabad in southern India. A total of 2522 subjects of all ages, who were representative of the Hyderabad population, participated in the population-based Andhra Pradesh Eye Disease Study. Of these subjects, 1859 aged >15 years responded to a structured questionnaire on cataract, glaucoma, night blindness and diabetic retinopathy to trained field

investigators. Having heard of the eye disease in question was defined as awareness and having some understanding of the eye disease was defined as knowledge. Awareness of cataract (69.8%) and night blindness (60.0%) was moderate but that of diabetic retinopathy (27.0%) was low, while that of glaucoma (2.3%) was very poor. Knowledge of all the eye diseases assessed was poor. Subjects aged > 30 years were significantly more aware of all eye diseases assessed except night blindness. Multivariate analysis revealed that women were significantly less aware of night blindness (odds ratio (OR) = 0.78; 95% confidence interval (CI) = 0.63–0.97). Education played a significant role in awareness of these eye diseases. Study subjects of upper socioeconomic status were significantly more aware of night blindness (OR = 2.20; 95% CI = 1.29–3.74) and those belonging to upper and middle socioeconomic strata were significantly more aware of diabetic retinopathy (OR = 2.79; 95% CI = 2.19–3.56). Muslims were significantly more aware of cataract (OR = 2.36; 95% CI = 1.84–3.02) and less aware of night blindness (OR = 0.52; 95% CI = 0.42–0.64). The major source of awareness of the eye diseases was a family member/friend/relative suffering from that eye disease.

These data suggest that there is a need for health education in this Indian population to increase their level of awareness and knowledge of common eye diseases. Such awareness and knowledge could lead to better understanding and acceptance of the importance of routine eye examinations for the early detection and treatment of eye diseases, thereby reducing visual impairment in this population.

2.3 Open-angle glaucoma in an urban population in southern India (Srinivas *et al.*, 2000).

The objective of this study was to assess the prevalence and features of open-angle glaucoma in an urban population in southern India. A total of 2522 persons (85.4% of those eligible) of all ages, including 1399 persons 30 years of age or older, from 24 clusters representative of the population of Hyderabad city.

Definite POAG, suspected POAG, and OHT were present in 27, 14, and 7 participants, respectively, with age- and gender-adjusted prevalence (95% confidence interval) of 1.62%

(0.77%–2.48%), 0.79% (0.39%–1.41%), and 0.32% (0.10%–0.78%) in those 30 years of age or older, and 2.56% (1.22%–3.91%), 1.11% (0.43%–1.78%), and 0.42% (0.11%–1.12%) in those 40 years of age or older, respectively. The prevalence of POAG increased significantly with age using multivariate analysis ($P < 0.001$). Only two of 27 participants (7.4%) with definite POAG had been previously diagnosed and treated, and 66.7% of the previously undiagnosed had IOP less than 22 mmHg. Fourteen of 27 participants (51.9%) with definite POAG had severe glaucomatous damage based on optic disc and visual field criteria, of which five participants (18.5%) had at least one blind eye as a result of POAG (all with best-corrected distance visual acuity less than 20/400 or central visual field less than 10°); the other 13 participants (48.1%) had moderate glaucomatous damage. Because visual fields and optic disc photography were not performed on all participants, the prevalence of POAG may have been underestimated. Secondary open-angle glaucoma was present in one participant as a result of angle recession.

The prevalence of open-angle glaucoma in this urban population in southern India is at least as much as that reported recently from white populations in developed countries. However, the vast majority of persons with glaucoma were undiagnosed in this population, and a large proportion of those having definite POAG already had severe glaucomatous damage.

2.4 Determinants of glaucoma awareness in a general eye clinic (Alice *et al*., 2000).

Purpose of this study was heightened public awareness about glaucoma may increase the chance of identifying undetected cases. To ascertain determinants of glaucoma awareness, we surveyed a population visiting a general eye clinic. 1197 general eye clinic patients and their companions were the participants. Glaucoma awareness overall (72%) approached that found in the subgroup self-reporting a diagnosis of glaucoma (80%). Survey attributes associated with an increased likelihood of being unaware of glaucoma were African American race (OR = 1.69 [1.28–2.20], Hispanic ethnicity (OR = 2.13 [1.46–3.02]), and less than a college education (OR = 1.67 [1.37–2.05]). Age was also a determinant of glaucoma awareness (for ages 50–64 years, OR = 0.60 [0.44–0.80] and for ages 65–79 years, OR = 0.56 [0.41–0.75] compared with ages less

than 35 years). A self-report of glaucoma was not a determinant of glaucoma awareness (OR = 0.63 [0.33–1.17]), although there was a trend toward enhanced glaucoma awareness in this subgroup. Finally, respondents with a history of employment in the health field (OR = 0.63 [0.49–0.82]) myopia (OR = 0.68 [0.56–0.82]), glaucoma in a first-degree relative (OR = 0.68 [0.53–0.87]), and respondents who reported having a dilated eye examination (OR = 0.53 [0.42–0.66]) were less likely to be unaware of glaucoma than those who did not have these attributes.

Although glaucoma awareness in this population was high, Hispanics, African Americans, and those with less than a college education were more likely to be unfamiliar with the disease. Interestingly, a self-report of having glaucoma was not a statistically significant determinant of glaucoma awareness.

2.5 Utilization of eye care services in an urban population in southern India: the Andhra Pradesh eye disease study (Dandona *et al*., 1999).

Aim of the study was to assess utilization of eye care services by people with visual impairment <6/18 or equivalent visual field loss in the better eye in the urban population of Hyderabad in southern India. 2522 subjects of all ages, representative of the population of Hyderabad city, underwent a detailed interview and dilated examination as part of the population based Andhra Pradesh eye disease study. Subjects more than 15 years of age were interviewed regarding the use of eye care services.

RESULTS Of 250 subjects with presenting distance visual acuity <6/18 or equivalent visual field loss in the better eye, information on utilization of eye care services was available for 229 (91.6%). Of these 229 subjects, 44 (19%) had visual acuity <6/60 or equivalent visual field loss in the better eye, and 202 (88.2%) had noticed decrease in vision over the past 5 years. Multivariate analysis showed that this decrease in vision was noticed significantly less by subjects with refractive error as the cause of visual impairment (odds ratio 0.34, 95% confidence interval 0.12–0.93). Of the 229 subjects who were visually impaired, 108 (59%) did not seek treatment. Multivariate analysis revealed that the odds for seeking treatment were

significantly lower for Hindus than Muslims (odds ratio 0.53, 95% confidence interval 0.28–0.98). The reasons for not seeking treatment could be classified as personal (49.5%), economic (30.8%), and social (19.6%).

A large proportion of subjects with visual impairment in this urban population in India did not seek treatment even after noticing decrease in vision. Projecting these data to the 155 million urban population >15 years of age in India, there may be 4.9 million (95% confidence interval 4.3–5.5 million) people in urban India who are not seeking treatment for their visual impairment even after noticing decrease in vision. These data suggest that efforts have to be made to better understand the reasons for this phenomenon so that optimal utilization of the available eye care services in urban India can be planned.

2.6 A study on the awareness of cataract disease and treatment options in patients who need surgery in a rural area of Eastern China (Zhou *et al*., 2008).

Purpose of the study was to investigate the awareness of cataract disease and treatment and to determine the major barriers for patients who need cataract treatment in a rural area of eastern China. A total of 251 cataract patients were selected by means of eye disease screenings throughout Jiangyan County. Questionnaires were administered after the doctor determined that the patient needed cataract surgery. A total of 89.6% of patients had been aware of their condition for more than 1 year. Only 49.8% of all patients had known for more than 1 year that their eye disease could be treated. The major barriers for those seeking eye treatments included residual functional vision (49.0%), financial problems (36.7%), no demand for the operation (8.8%), and skepticism about the operation (8.8%). Poor vision function grade and female gender were two significant factors associated with a longer awareness (>3 years) of the existence of cataracts. Patients with a history of eye disease and a longer awareness of eye disease were more likely to have known about the potential treatments for a longer period of time (>1 year).The patients' awareness of the presence of cataract disease and potential treatment were unbalanced. The main treatment barriers were lower demand for visionimprovement and financial problems. It is imperative to

educate patients on eye health care and to provide low cost, but high quality, cataract surgery to these patients.

2.7 Chronic open angle glaucoma: patient awareness of the nature of the disease, topical medication, compliance and the prevalence of systemic symptoms (Deokule *et al*., 2003).

Purpose of the study was to study the awareness of the nature of the disease, compliance with treatment, and prevalence of systemic symptoms in a group of patients with chronic open angle glaucoma (COAG).

A structured questionnaire was designed and given to 260 consecutive COAG patients attending a general ophthalmology clinic. Questions related to the increased risk of COAG amongst family members, screening of family members, nature of field defects, variation in IOP, topical treatment and availability of a free eye test for a COAG patient in the UK were asked. Compliance and systemic symptoms were also assessed. Forty-one per cent (107 of 260) of patients in the study group were aware of the increased risk of COAG in family members and 45.5% (118 of 260) of patient's family members had undergone screening for COAG. Seventy-three per cent (191 of 260) of the patients were aware of their own and their family members' entitlement to a free eye test. Seventy-seven per cent of patients claimed full compliance. Thirty per cent of patients were noted to have systemic symptoms. The awareness of the nature of COAG in this population was low and incidence of perceived drug related systemic symptoms very high. Both of these may contribute to poor compliance.

2.8 Factors Associated with Awareness, Attitudes and Practices Regarding Common Eye Diseases in the General Population in a Rural District in Bangladesh: The Bangladesh Population-based Diabetes and Eye Study (Islam *et al*., 2015).

Purpose of this study was to assess the awareness, attitudes, and practices associated with common eye diseases and eye care utilization in a rural district of Bangladesh. Data were collected using a multilevel cluster random sampling technique from 3104 adults aged ≥30

years from the Banshgram union with a questionnaire assessing the awareness, attitudes and practice about diabetes and common eye diseases, educational attainment, socio-economic status, and medical history.

Participants were aged between 30 and 89 years with a mean (SD) age of 51 (12) years and 65% were female. The majority of participants had heard of cataracts (90%), trachoma (86%) and Pterygium (84%), yet only 4% had heard of diabetic retinopathy (DR), 7% of glaucoma and 8% of Age-related macular degeneration (AMD). However, 58% of participants did not know vision loss could be prevented. Factors associated with lower awareness regarding common eye diseases were increasing age, lack of formal schooling, and lower socio-economic status. A lower proportion (57%) of people with no schooling compared to those who had attained at least secondary school certificate education (72%) reported that they knew that vision loss could be prevented ($p<0.001$). Overall 51% of people had heard of at least six (67%) out of nine items relating to awareness of common eye diseases. This included 41% of participants aged 65 years or older compared to 61% of those aged 30–35 years ($p<0.001$). Only 4% had an eye check at least once a year and higher education and better SES were associated with higher frequency of eye checks.

In rural Bangladesh awareness of cataract, trachoma and pterygium was good but limited in relation to the potentially blinding conditions of glaucoma, DR, and AMD. The results show a large gap between public awareness and treatment practices about common eye diseases. Public health promotion should be designed to address these knowledge gaps.

2.9 Impact of Dry Eye Syndrome on Vision-Related Quality of Life in a Non-Clinic-Based General Population (Qihua *et al.*, 2012).

Purpose of this study was Dry eye syndrome (DES) is a common ocular disorder occurring in general population. The purpose of this study is to evaluate the impact of DES on vision-related quality of life (QoL) in a non-clinic-based general population.

A total of 229 participants were enrolled in the study, with an average age of (60.7 ±10.1) years old. Majority of these participants were female (59.8 %, 137/229). The total DES symptom scores (TDSS) in subjects either with definite DES or only with dry eye symptoms were significantly higher ($F = 60.331$, $P < 0.001$). The values of tear break-up time (TBUT) and Schirmer test were significantly lower in participants with DES and those with dry eye signs only ($F = 55.158$ and 40.778, $P < 0.001$). The composite score of the NEI VFQ-25 was significantly lower in subjects with DES ($F = 4.901$, $P = 0.003$). Moreover, the subscale scores of ocular pain and mental health were significantly lower in those with either DES or dry eye symptoms only ($F = 10.962$ and 7.362 respectively, both $P < 0.001$). The multiple regression analysis showed that the TDSS had a significant negative correlation with the VFQ-25 composite score as well as with the subscale score for ocular pain and mental health, even after the adjustment of all other factors (all $P < 0.01$).

The symptoms of dry eye are associated with an adverse impact on vision-related QoL in non-clinic-based general population, which is mainly represented as more ocular pain and discomfort, and impaired mental health as well. Apart from clinical examination, it is also important to refer to subjective symptoms and QoL scores when assessing the severity of DES.

2.10 The age-related eye disease study system for classifying age-related macular degeneration from stereoscopic color fundus photographs: the age-related eye disease study report (American Journal of Ophthalmology, 2011).

Purpose of this study was to describe the system for grading age-related macular degeneration from fundus photographs in the Age-Related Eye Disease Study. This is a prospective multicenter cohort study of the course of age-related macular degeneration and a placebo-controlled clinical trial of the effect of high-dose vitamin and mineral supplements on development of advanced age-related macular degeneration.

In a cumulative sample of 1230 eyes for contemporaneous reproducibility, agreement on advanced age-related macular degeneration was 96% (kappa = 0.88) and for four-step age-related macular degeneration level was 83% ($\kappa = 0.77$). Agreement was moderate for pigment

epithelial detachment (κ = 0.54) and substantial for serous sensory retinal detachment, hard exudates, subretinal/subretinal pigment epithelial hemorrhage, subretinal fibrous tissue, and central geographic atrophy (κ = 0.73–0.82). For pigment abnormalities, agreement was substantial for geographic atrophy (κ = 0.63; $\kappa_{weighted}$ = 0.71, 0.75 weight for one-step disagreements), and moderate for depigmentation (κ = 0.41, $\kappa_{weighted}$ = 0.51) and increased pigment (κ = 0.54, $\kappa_{weighted}$ = 0.71). For drusen, agreement was moderate to substantial for presence/maximum size (κ = 0.50, $\kappa_{weighted}$ = 0.68), type (κ = 0.61, $\kappa_{weighted}$ = 0.69), and area (κ = 0.56, $\kappa_{weighted}$ = 0.77). Six annual regrades of 119 eyes showed little temporal drift in grading of various age-related macular degeneration abnormalities, except for drusen type.

The Age-Related Eye Disease Study has demonstrated satisfactory reliability for detecting onset of advanced age-related macular degeneration in a cohort, and moderate to substantial agreement on various abnormalities across the age-related macular degeneration spectrum. The Age-Related Eye Disease Study system for classification of age-related macular degeneration is suitable for longitudinal multicenter studies.

Significance of the study

Eye-care services in Bangladesh are provided by the government, non-governmental organizations, and private sector. These services range from services free of cost to the economically underprivileged, to charges covering the costs relating to only the consumables in case of surgery, and to charges with profit margins. Most of the eye-care infrastructure is concentrated in the urban areas making access of these services difficult for those living in the rural parts of the country. The emphasis needs to be on developing reasonable-quality sustainable infrastructure in the underserved rural areas that will serve the eye-care needs of the population in the long-term. Glaucoma is one of the leading causes of irreversible blindness in developing nations. It is now increasingly being recognized as a major cause of ocular morbidity that requires urgent attention. Awareness of glaucoma in our rural study population was very poor compared to the urban population. Even among the rural people who were aware of the disease, knowledge about glaucoma was very poor. Limited access to medical and diagnostic care in the rural areas may have contributed to poor knowledge and awareness of eye diseases. Adequate access and proper utilization of eye care services can create greater awareness and exposure to information about various eye diseases including glaucoma. Though awareness of eye disease in the urban population was not very high, those who were aware had reasonable knowledge of the disease. Education and socioeconomic status played a significant role in the level of awareness of eye diseases in the population (WHO, 2001).

While many complicated eye diseases can be treated in hospitals, public awareness of vision care issues remains low. Effective eye health education may influence individuals to consider screening and eye care. This may lead to early detection of eye disease and prevent blindness. Educating the rural community on the consequences of delayed treatment of eye diseases will be an important first component in the promotion of preventive ophthalmic care. Mass media and word of mouth can be effective tools for generating awareness on regular and timely eye checkup. The strategy should be to increase awareness of eye disease in selected high-risk target audience among the population of Bangladesh (WHO, action plan 2006-2011).

Aim and objective of the study

Aim and objective of the study was to assess the level of awareness of eye diseases among the population of Bangladesh.

Chapter three

Methods and Materials

3.1 Type of the study

It was a survey based study

3.2 Place of the study

This study was done among the people of Bangladesh.

3.3 Study Population

In this study, 750 people were included. Both male and female were included in the study.

3.4 Study Period

Study period was from September, 2014 to December, 2015.

3.5 Data collection paper

A data collection paper was made and compiled all the information and data of the people in an organized manner.

3.6 Data analysis

All the data were collected properly and then checked. After that the collected data were entered into Microsoft Excel and then the result was shown in pie chart and calculated the percentage of the parameter of the awareness of eye disease and eye health.

Chapter four

Result

4.1 Gender distribution

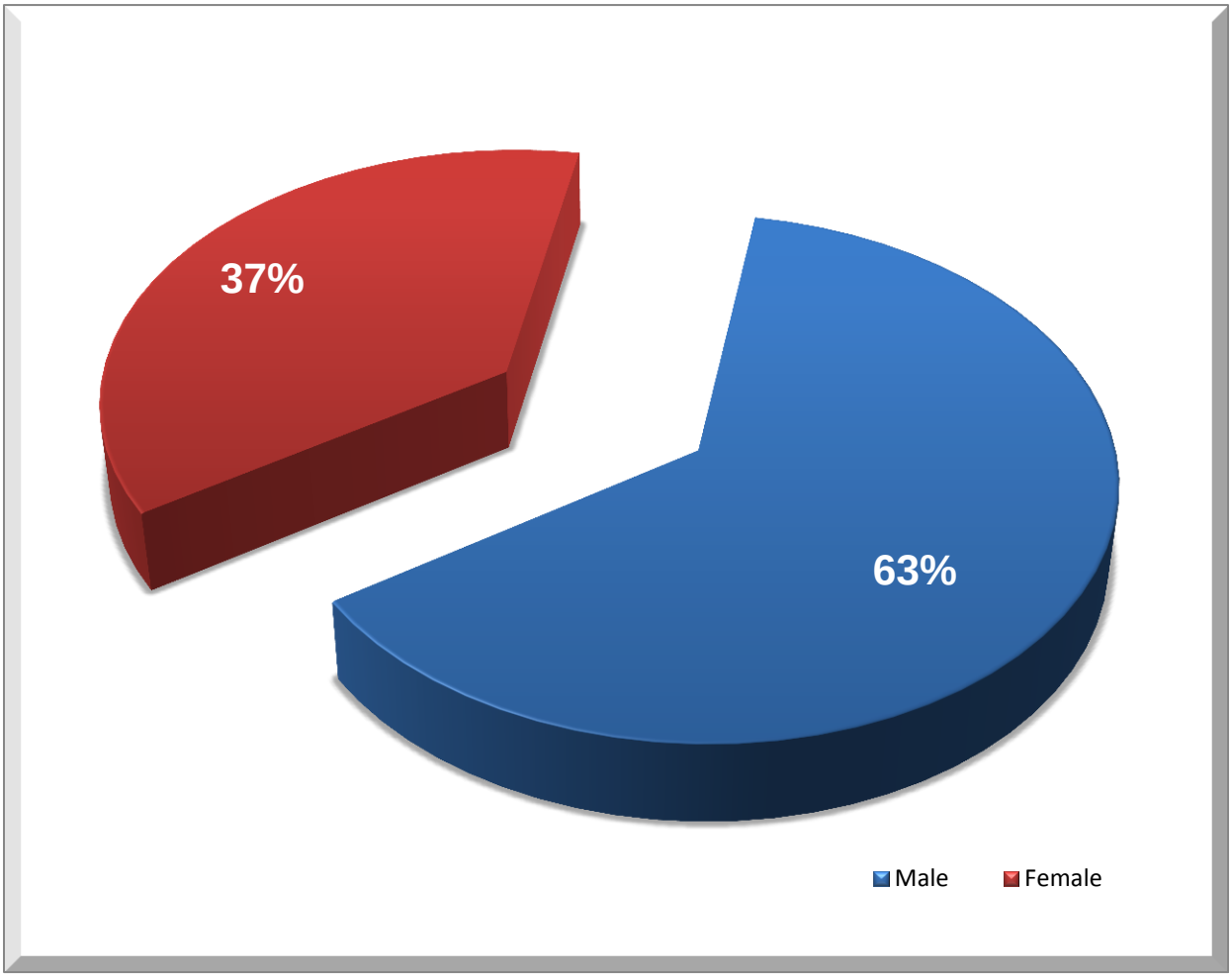


Figure 4.1 Gender Distribution

This diagram shows the gender distribution of the population, where 63% male and 37% female were included in this report. Different category people (e.g. urban, rural, slum etc.) were included in the study.

4.2 Age distribution

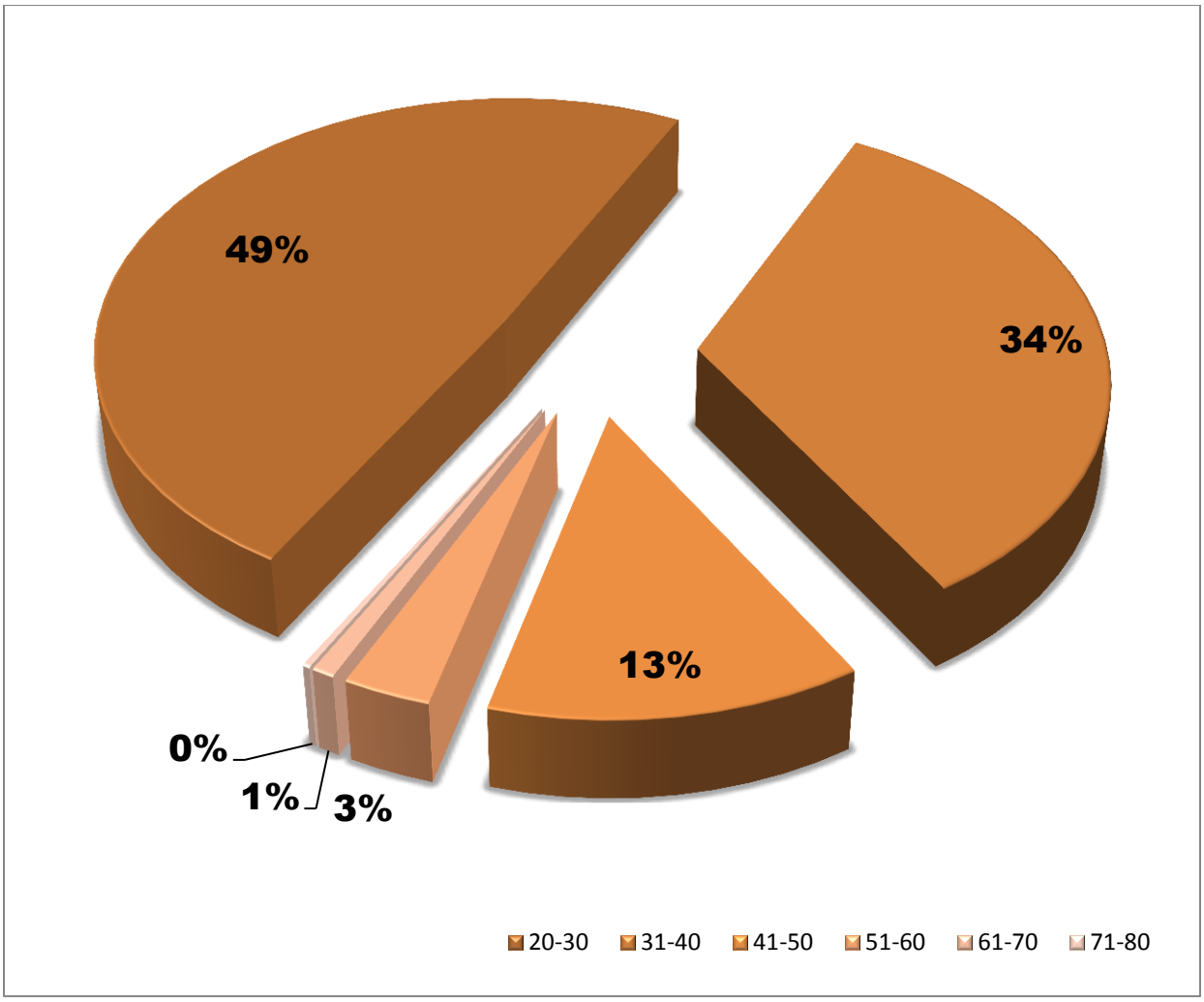


Figure 4.2 Age distribution

From this diagram we can see the age distribution of the population, where (20-30) 49%, (31-40)34%, (41-50) 13%, (51-60) 3%, (61-70) 1% and (71-80) aged people were 0% respectively.

4.3 Education level

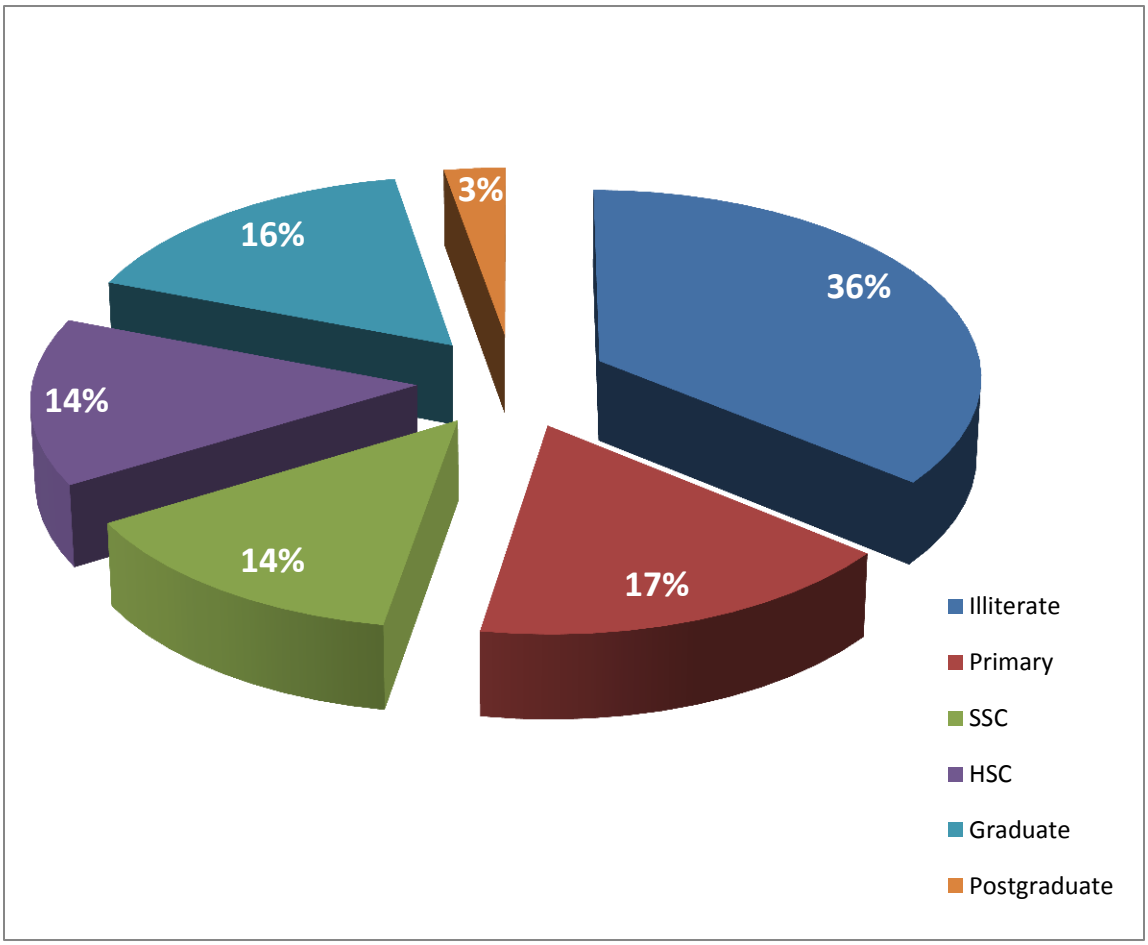


Figure 4.3 Education level

From this study we can see that educational level of the population, Where 36% were illiterate, 17% primary, 14% SSC, 14% HSC, 16% graduate and 3% postgraduate respectively.

4.4 Occupational level

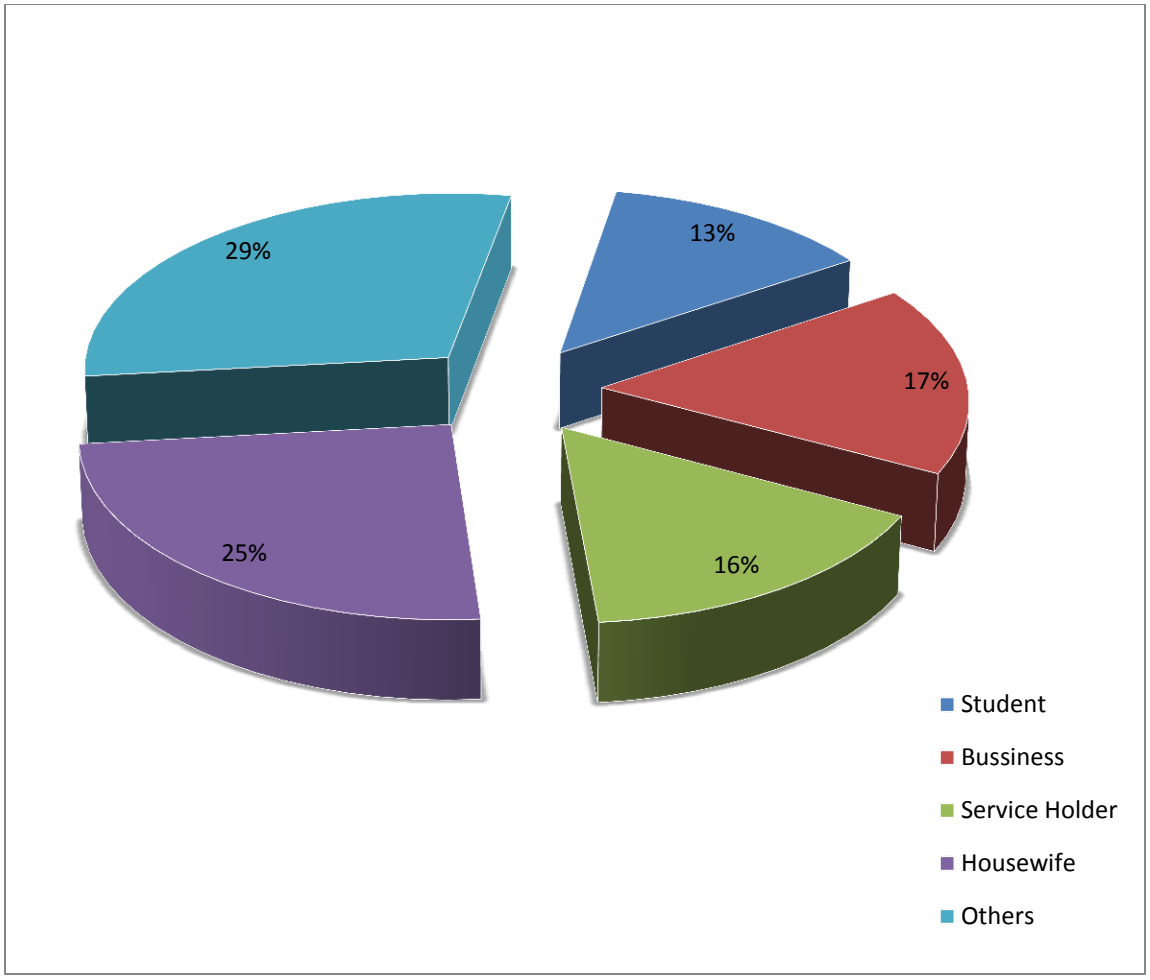


Figure 4.4 Occupational level

The occupational level of the population is describes through this diagram, Where 13% were student, 17% business man , 16% Service holder, 25% housewife, 29% others occupation respectively.

4.5 Stress on work

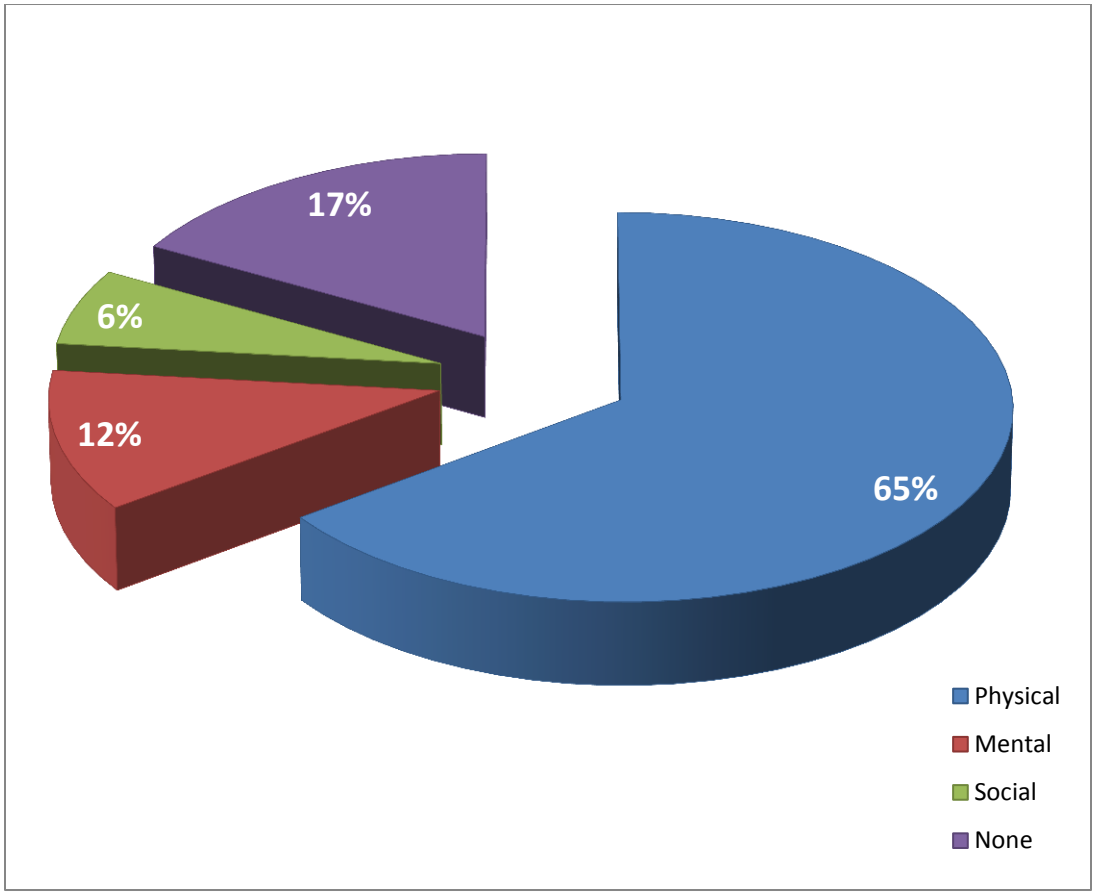


Figure 4.5 Stress on work

This diagram shows the stress on work of the population, Where 65% were physical, 12% mental, 6% social and 17% none respectively.

4.6 Smoking Habit

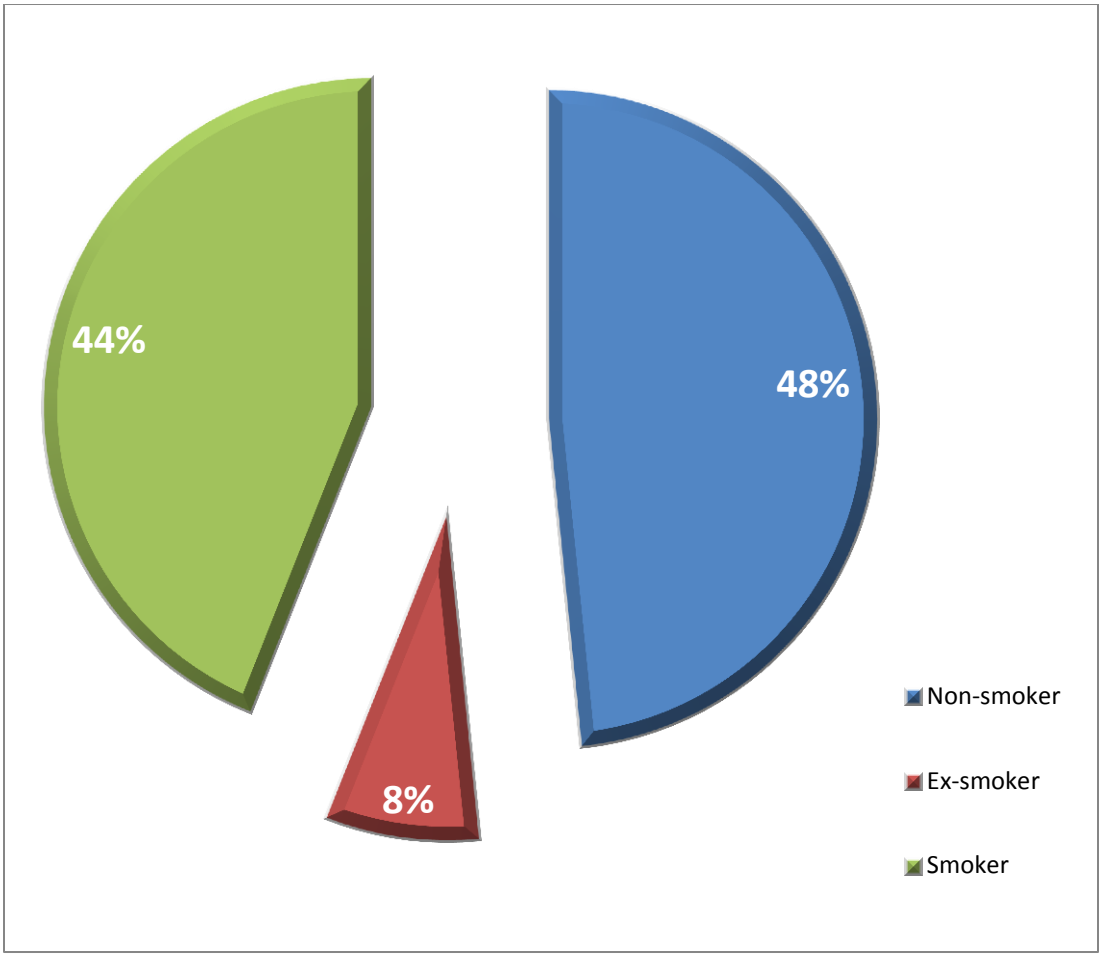


Figure 4.6 Smoking Habit

Through this diagram we can see the smoking habit of the population, Where 48% were non-smoker, 44% smoker and 8% ex-smoker respectively.

4.7 Regular eye checkup

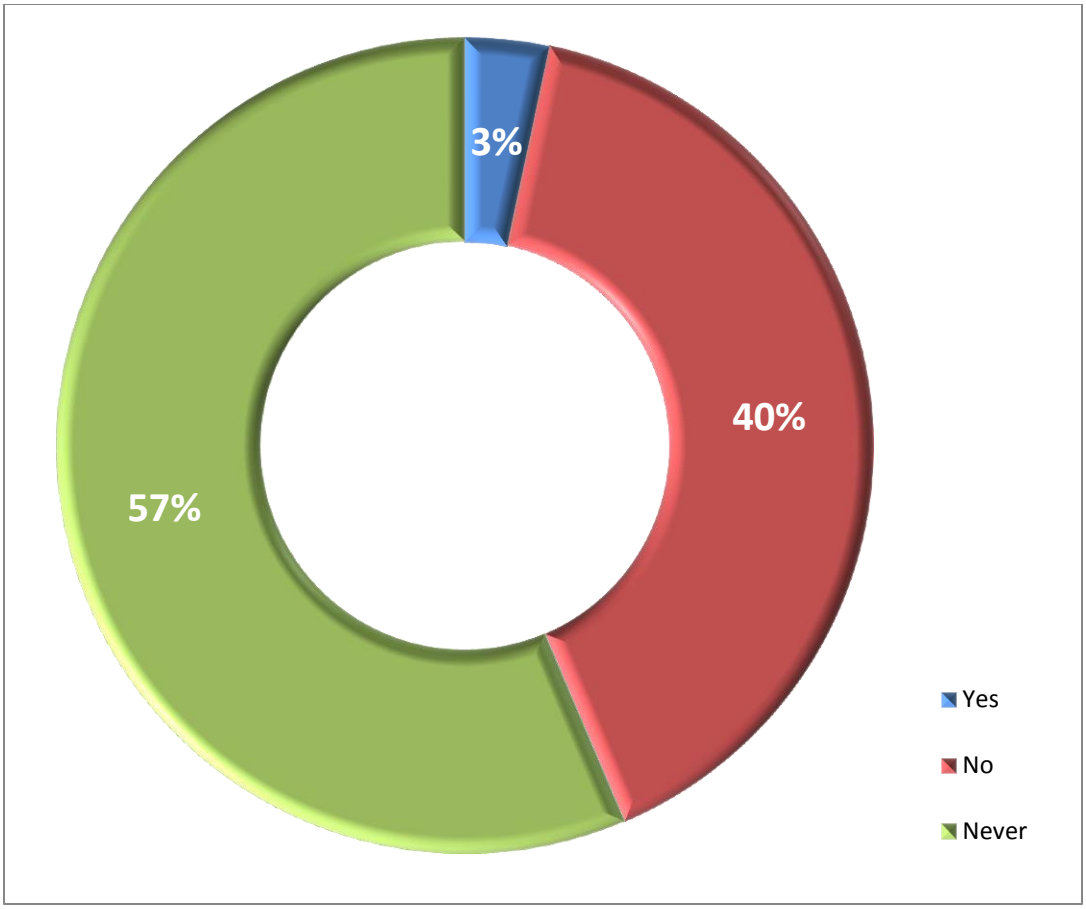


Figure 4.7 regular eye checkup

Only 3% people were properly concern about regular eye checkup and most of them (57%) had never check their eye.

4.8 Sleep duration

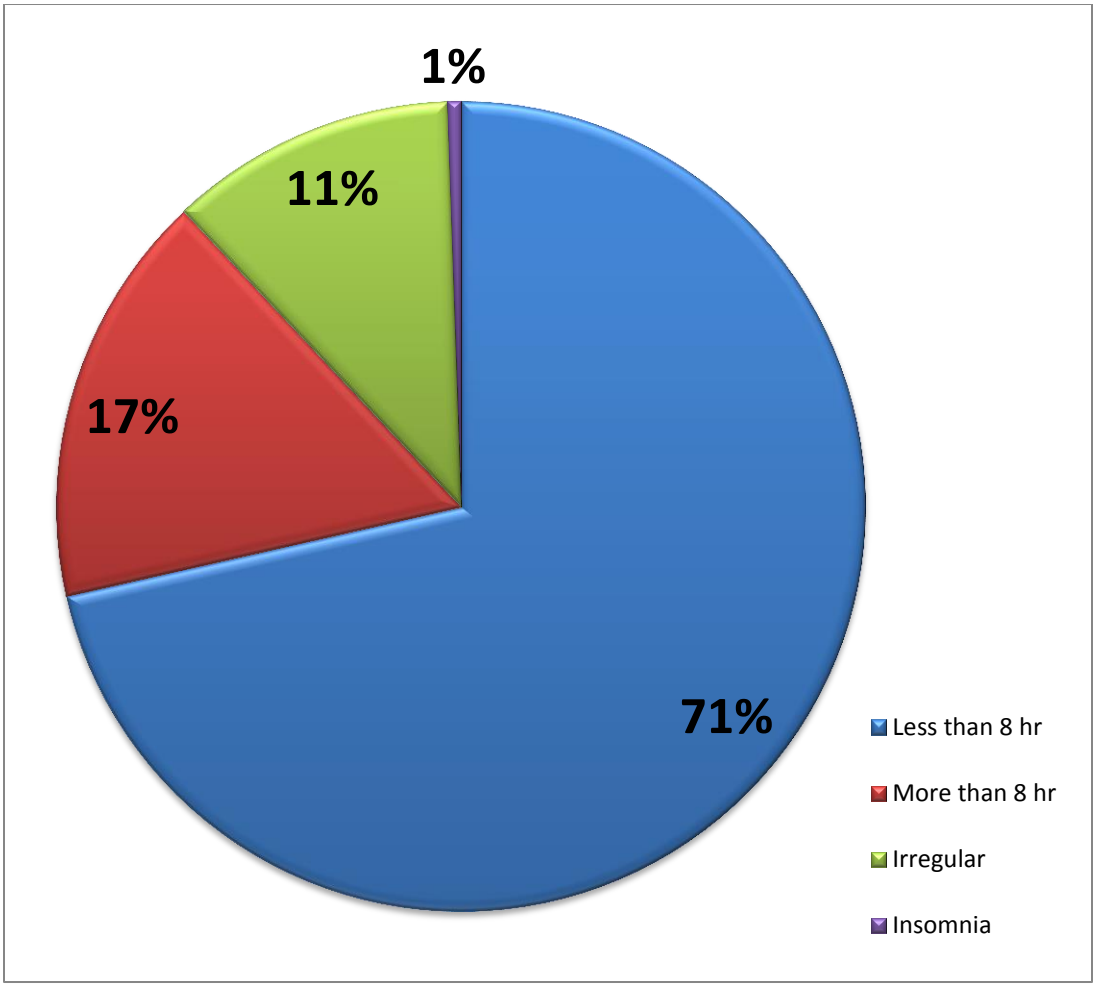


Figure 4.8 Sleep duration

These diagram shows the sleep duration of the population, Where 71% were sleep more than 8 hr, 17% less than 8 hr, 11% were irregular and 1% had Insomnia.

4.9 Diabetes

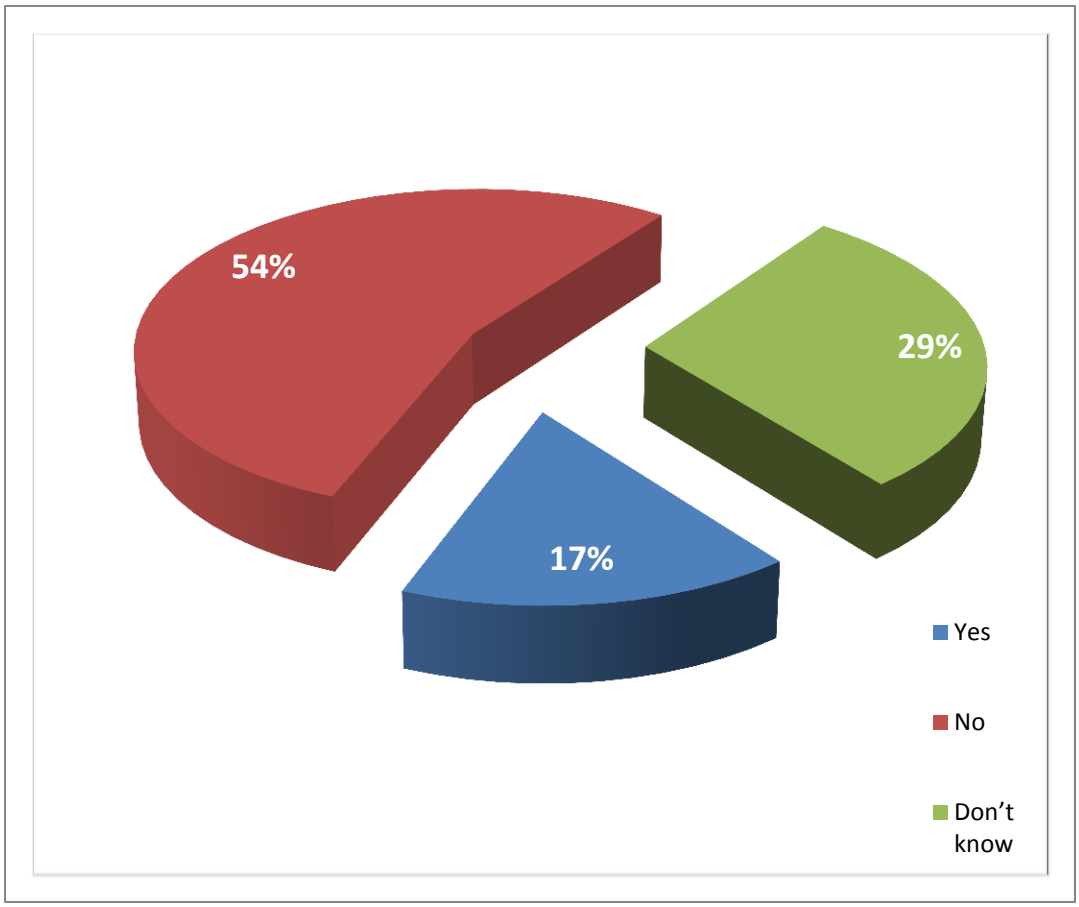


Figure 4.9 diabetes

Presence of diabetes among the population was 17% and large amount of the population (54%) totally don't concern of diabetes.

4.10 Awareness of Glaucoma

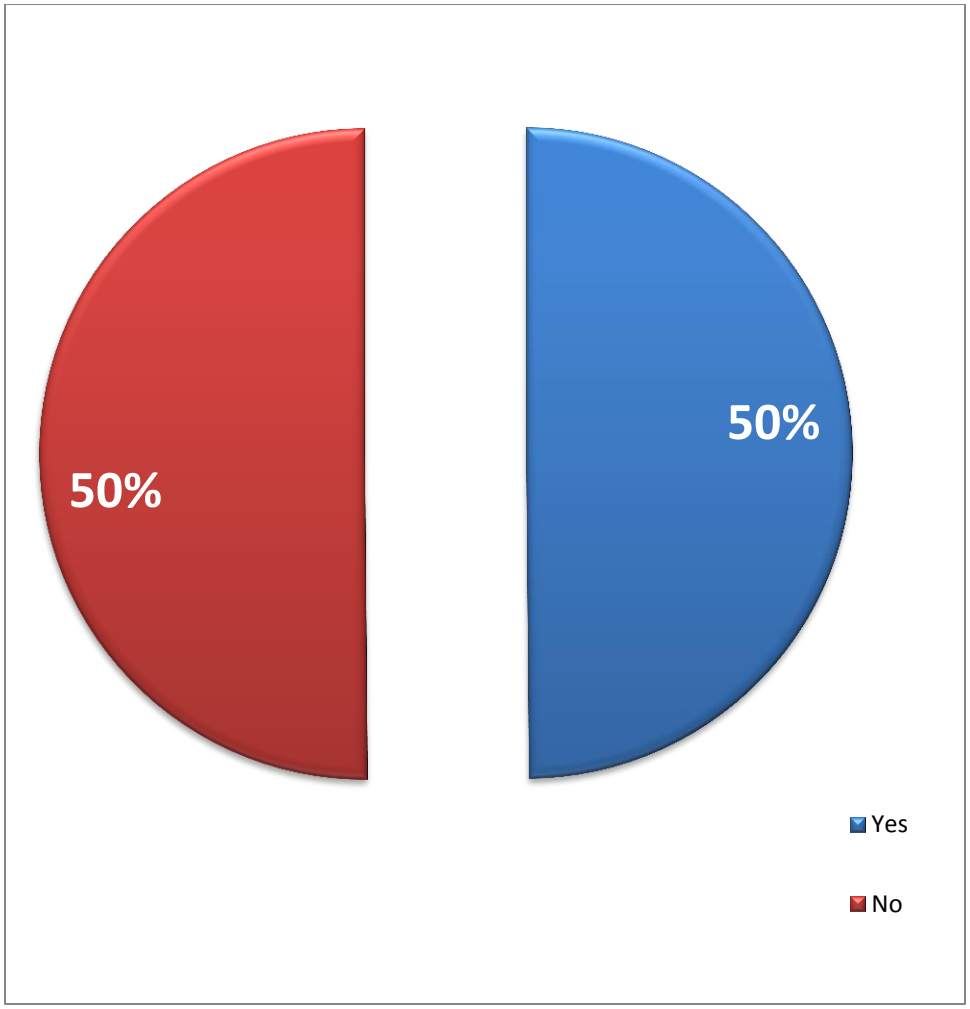


Figure 4.10 Awareness of Glaucoma

This survey based study shows that 50% of population were aware of Glaucoma 50% were not. From this result we can say that awareness of glaucoma among the population of Bangladesh is not sufficient.

4.11 Knowledge of Glaucoma

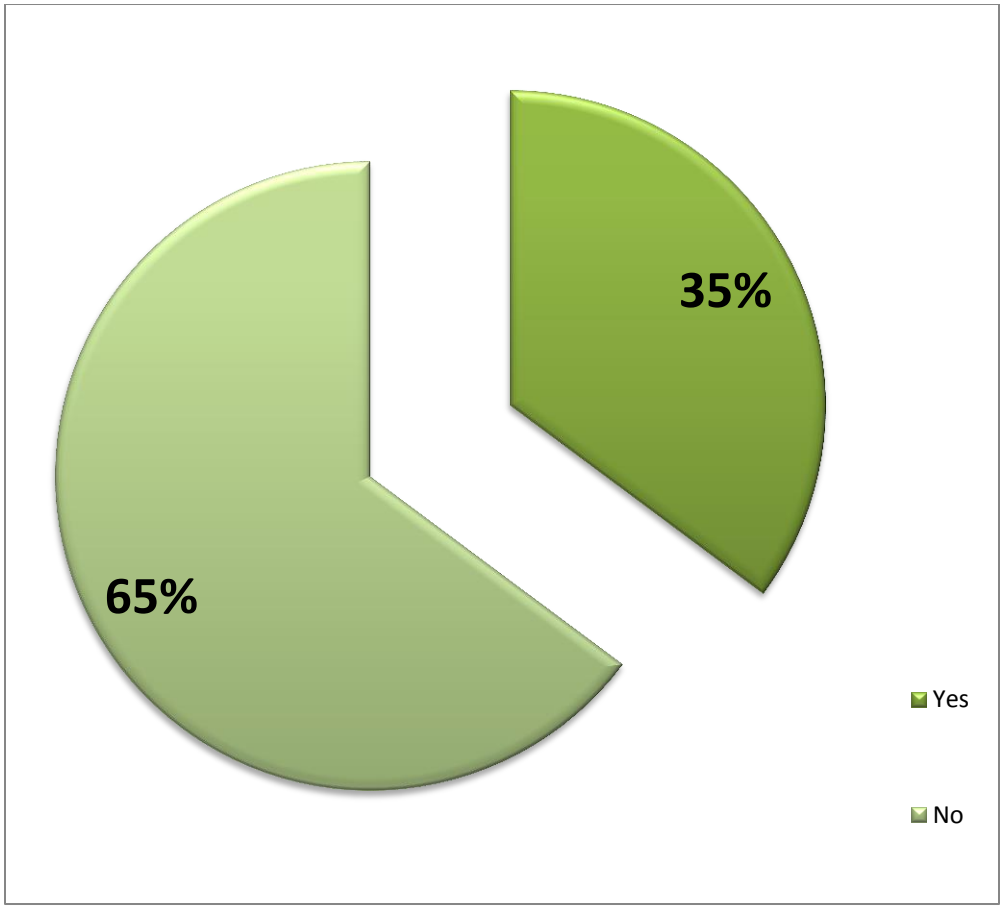


Figure 4.11 Knowledge of glaucoma

Knowledge level of glaucoma was very low among the population. Only 35% people had the knowledge of glaucoma.

4.12 Awareness of cataract

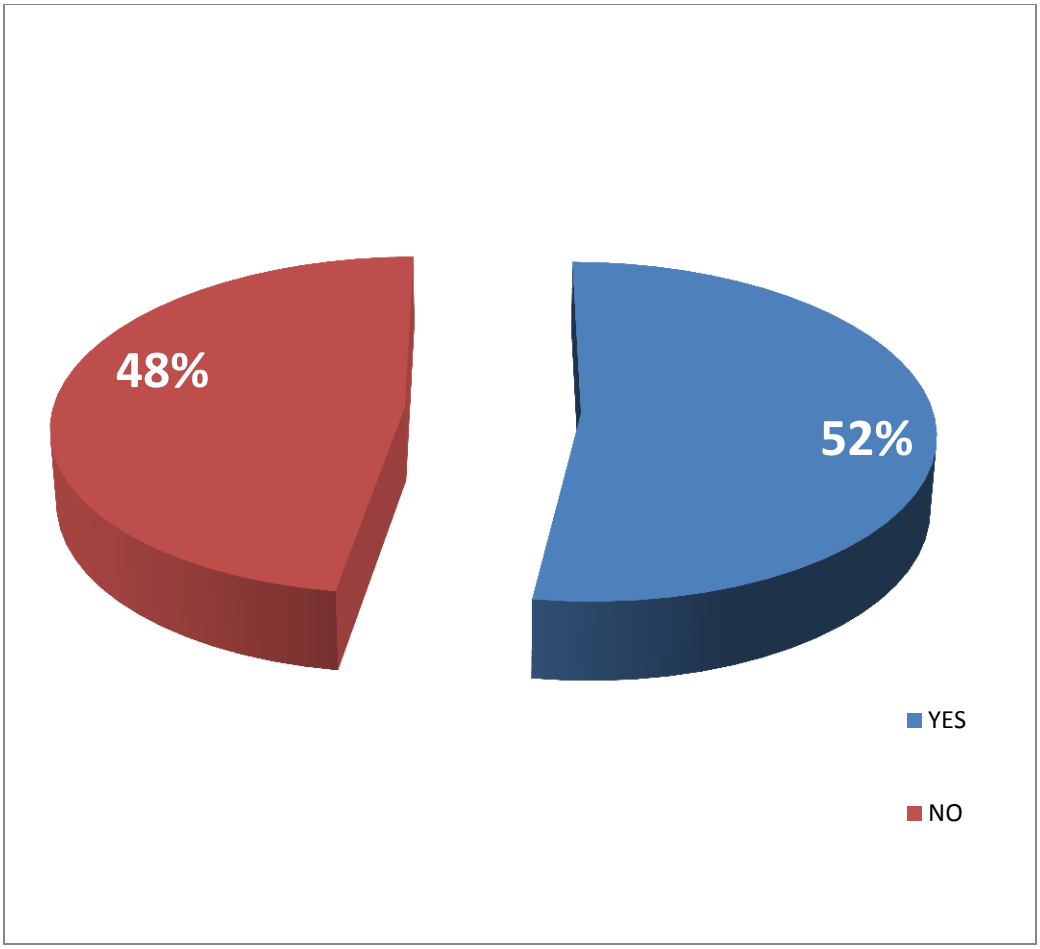


Figure 4.12 Awareness of cataract

According to this diagram we can see that the awareness of cataract belongs to 52% among the population, where 48% were not concern of cataract.

4.13 Knowledge of Cataract

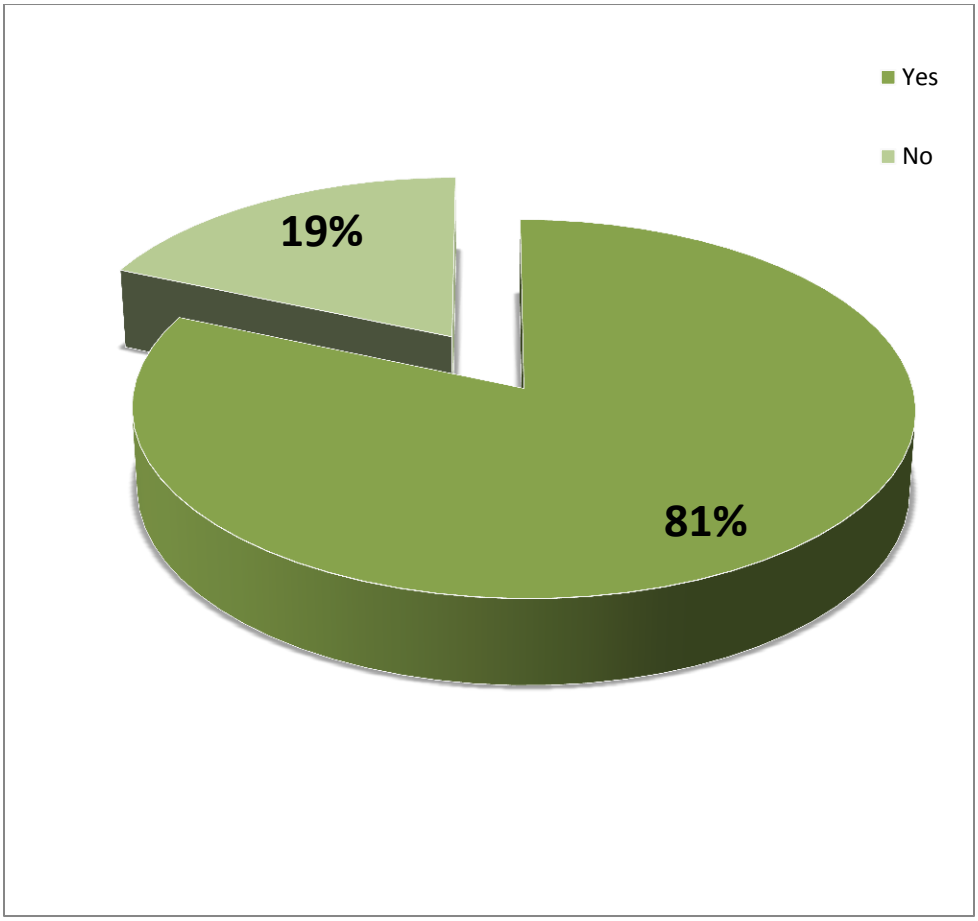


Figure 4.13 Knowledge of Cataract

The level of knowledge of cataract was tremendously low among the population. Only 19% people had the proper knowledge of cataract.

4.14 Knowledge of Night blindness

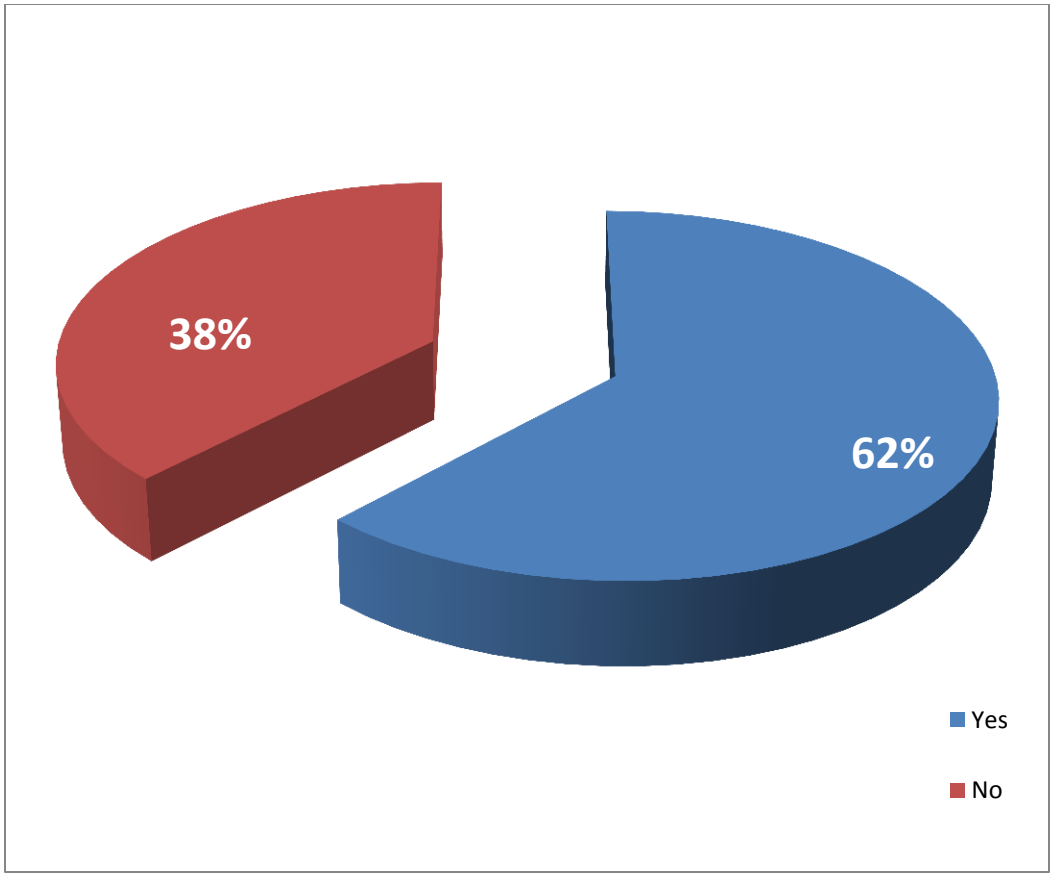


Figure 4.14 Knowledge of Night blindness

This diagram shows the knowledge level of the population about night blindness, where 38% people had the knowledge of night blindness and the ratio is not sufficient enough.

4.15 Knowledge about the interaction between diabetes and eye disease

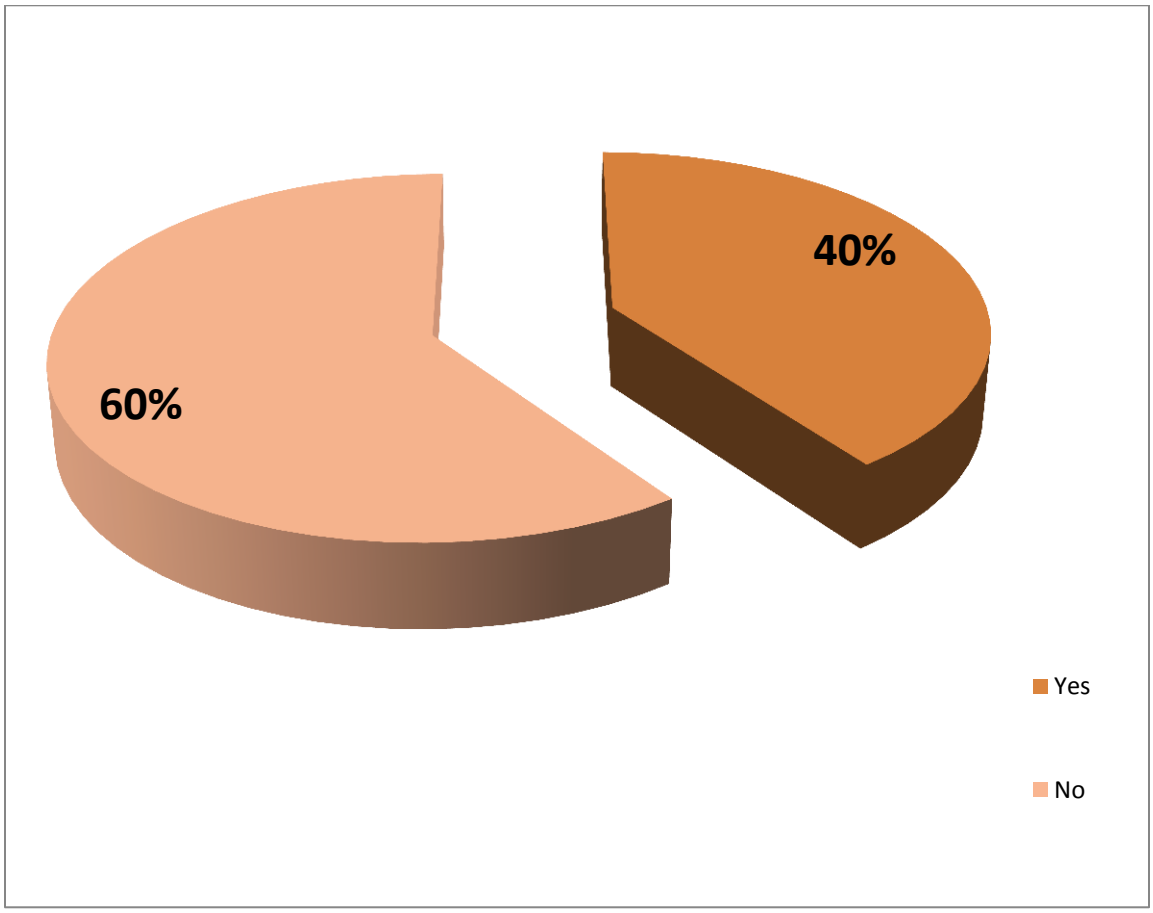


Figure 4.15 Knowledge about the interaction between diabetes and eye disease

This diagram shows that only 40% of the population had the knowledge about the interaction between diabetes and eye disease.

4.16 Concern about family's present eye health condition

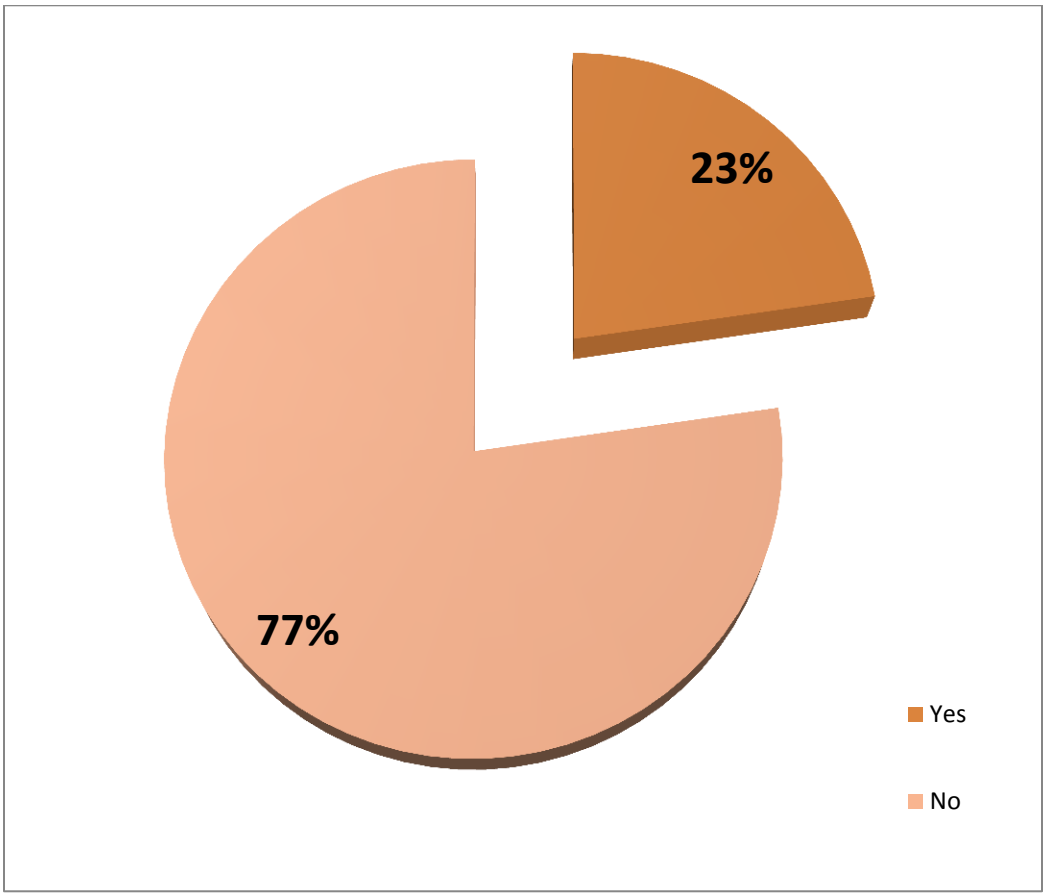


Figure 4.16 Concern about family's present eye health condition

From the result of this survey based study we can see that 77% people were not concern about their family's eye health condition. Lack of proper knowledge and socio economic state are remarkably responsible for these criteria.

4.17 Knowledge about Proper frequency of sight test

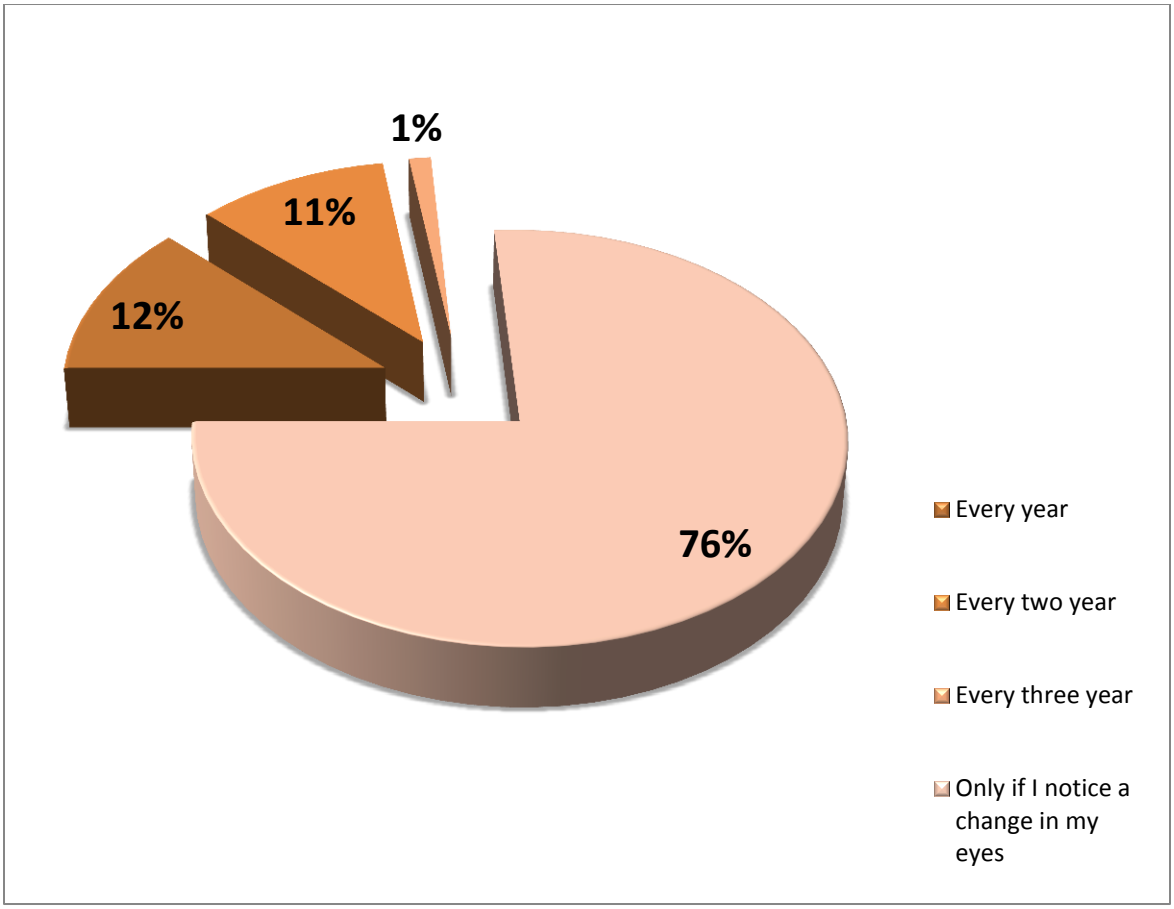


Figure 4.17 Knowledge about Proper frequency of sight test

Huge portion (76%) among the population doesn't know the proper frequency of sight test. Only 1% of total population have the proper knowledge and that is very much poor.

4.18 Time duration in front of TV/Computer/Mobile

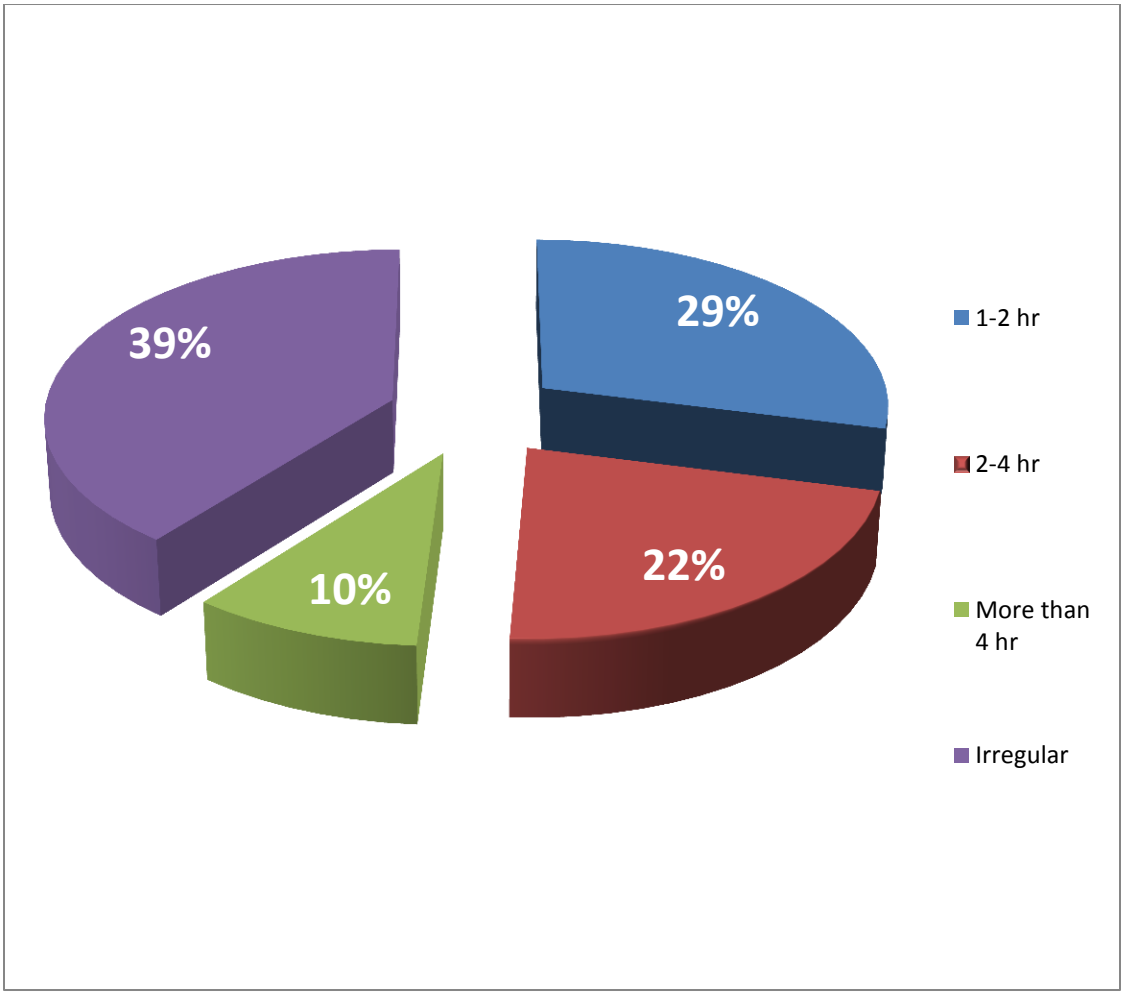


Figure 4.18 Time duration in front of TV/Computer/Mobile

This diagram shows the time duration in front of TV/Computer/Mobile of the population. Most of them don't know that these devices can cause serious eye problem.

4.19 Wearing sunglass for the protection of eye

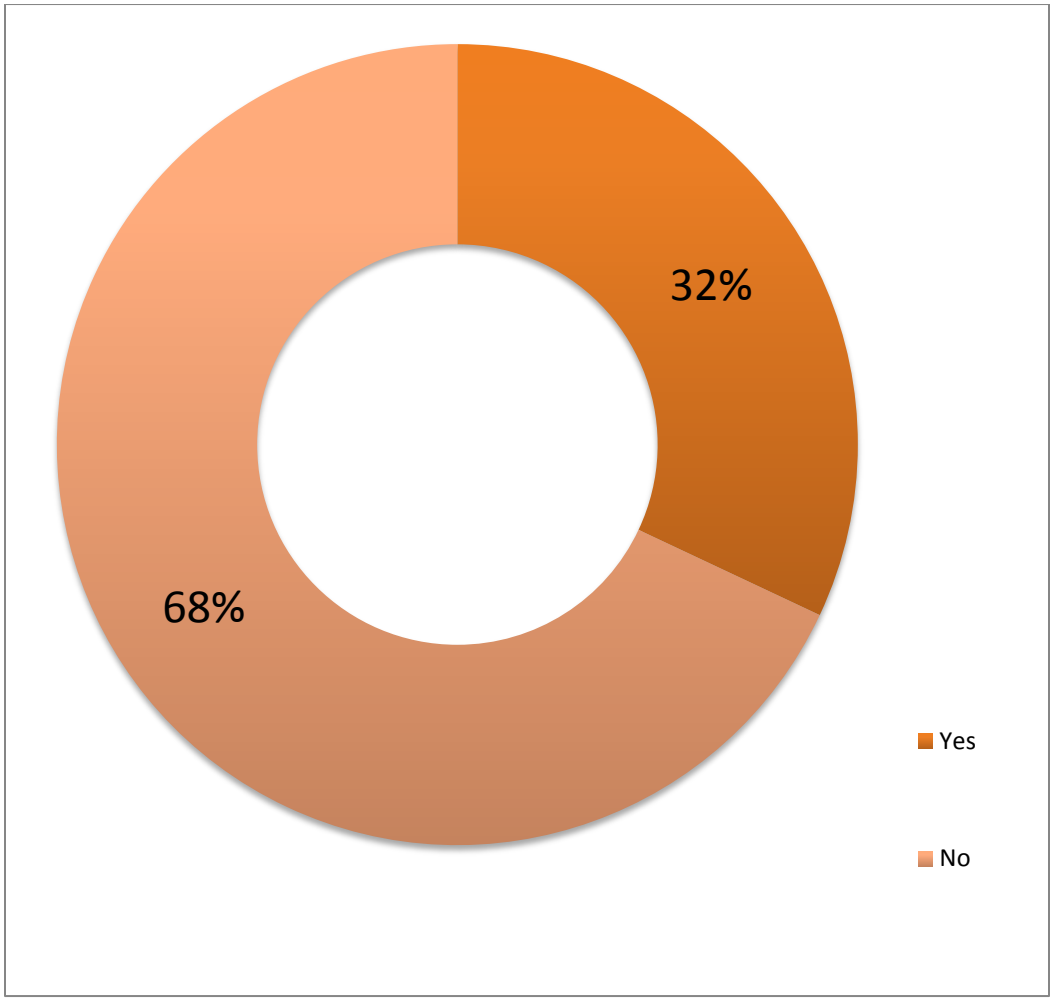


Figure 4.19 Percentage of Wearing sunglass for the protection of eye

Wearing sunglass is a good habit for the protection of eye and most of the people know it but they don't follow it. This diagram shows the percentage of the population of Wearing sunglass for the protection of eye. 68% population doesn't wear sunglass for the protection of their eye.

4.20 Frequency of taking nutritional food (colorful fruit, small fish, vegetables, fresh meat) for optimum eye health.

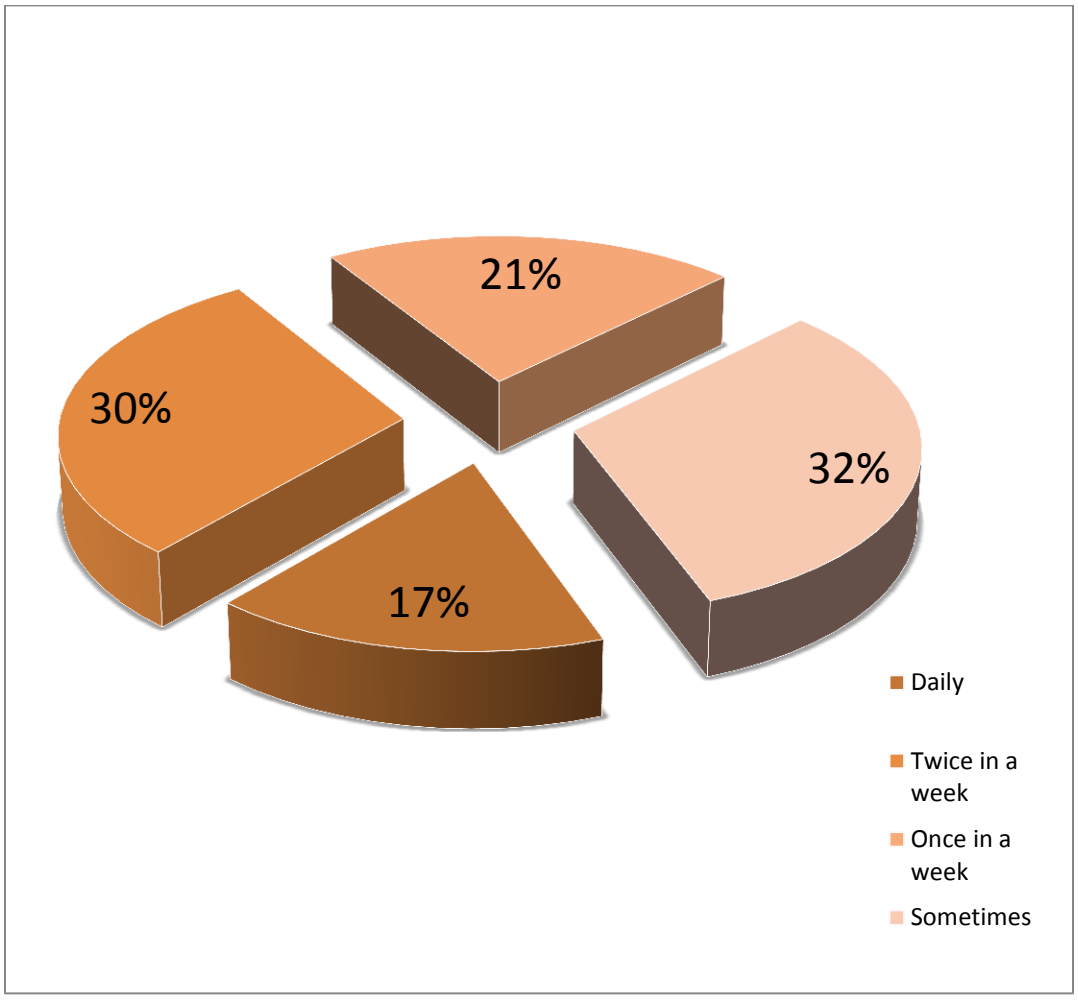


Figure 4.20 Frequency of taking nutritional food (colorful fruit, small fish, vegetables, fresh meat) for optimum eye health.

From this diagram we can see the frequency of taking nutritional food for optimum eye health. Only 17% people take those food daily and most of them sometimes. Lack of knowledge and awareness are responsible for these criteria.

4.21 Maintain blood pressure and obesity

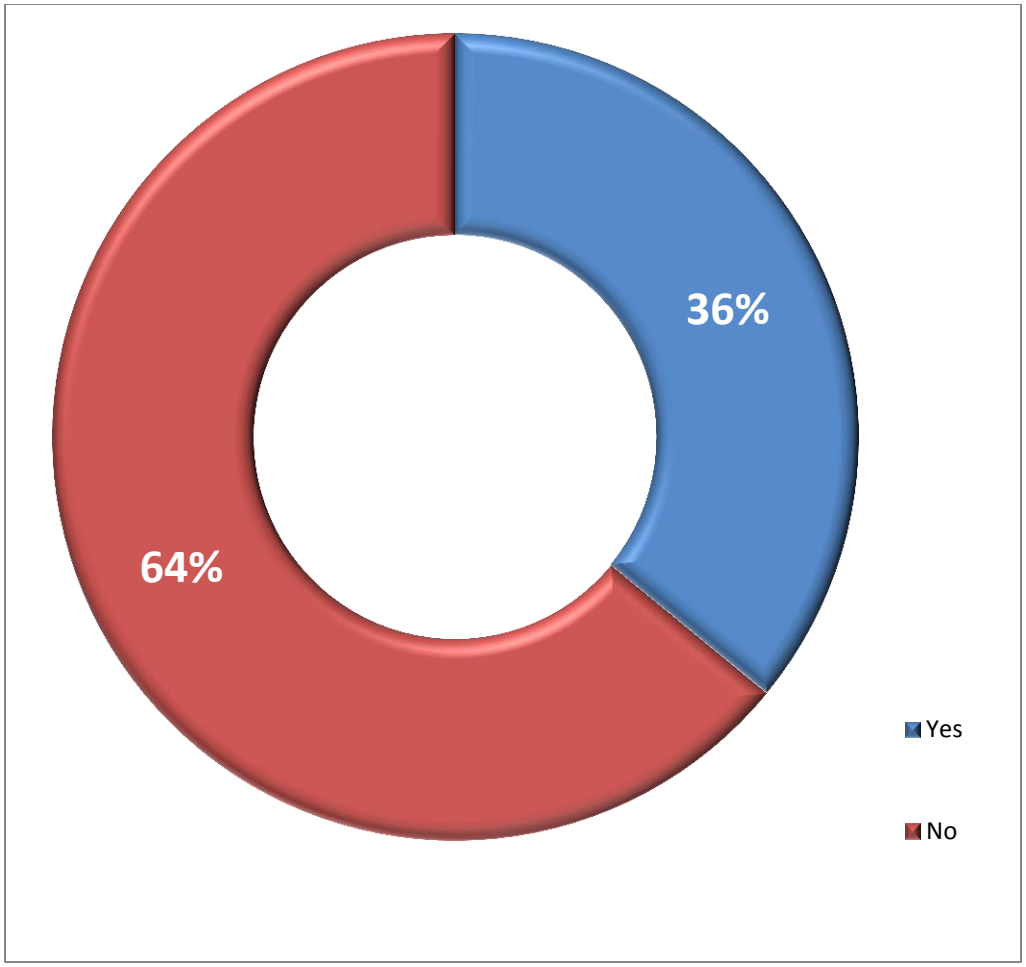


Figure 4.21 Maintain blood pressure and obesity

From this study it is significantly noticeable that only 36% of total population was maintaining their blood pressure and obesity. Most of them do not maintain their blood pressure and obesity because they don't have the knowledge.

4.22 Way of maintaining BP and obesity (103 people who maintain)

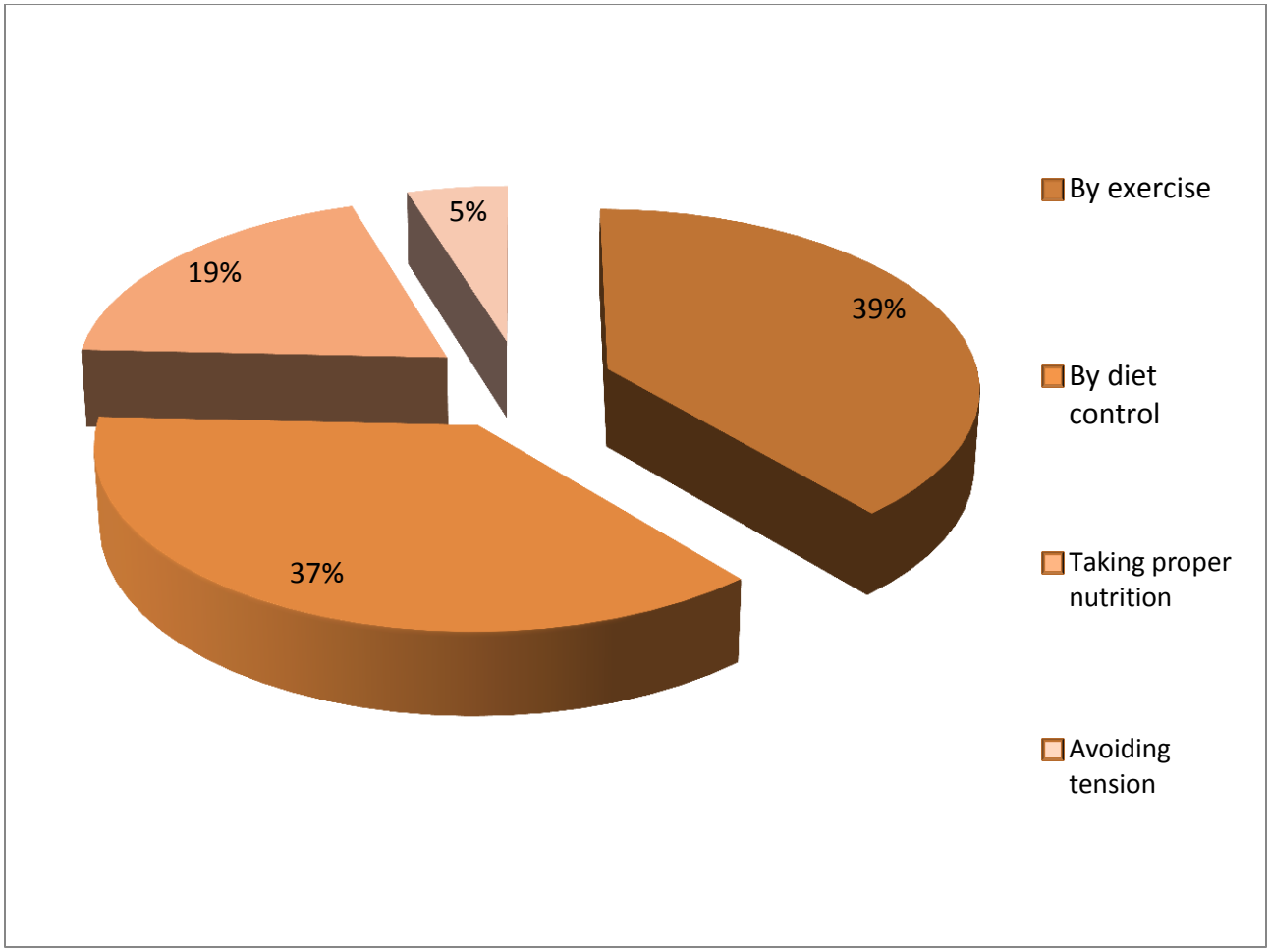


Figure 4.22 Way of maintaining BP and obesity (103 people who maintain)

Only 103 people maintain their blood pressure and obesity among 750 people. They maintain their blood pressure and obesity in different way. Most of them (39%) maintain it through exercise. 37% maintain it by controlling their diet. Others maintain it by taking proper nutrition and avoiding tension.

Chapter five

Discussion and Conclusion

5.1 Discussion

Awareness of glaucoma and cataract among the study population was poor. For glaucoma, early detection and prevention may prevent progression of the disease, but because of its "silent" nature early detection of glaucoma is difficult unless the patient undergoes an eye examination (WHO, 2001). Hence, an increase in the level of awareness of glaucoma in our population is essential if more people are to be screened for the condition. Even though the awareness of glaucoma was very poor, the finding of reasonable knowledge about it among the minority who were aware of the condition is encouraging. Most of the population of this survey knows that the risk of cataract increases by getting older. But most of them don't know the proper way how to maintain optimum eye health. From this study we can see that rate of elder people is much less than young people. Education is a big issue which can increase the knowledge and awareness among the population but proper knowledge is mandatory. From this study we saw that some educated people also were not properly concerned. Socio-economic development also needed for the improvement of public awareness. According to this study only 50% population were concern about glaucoma and 35% population had proper knowledge of glaucoma. Large number of study population had not enough knowledge of glaucoma and the percentage was 65%. Knowledge of cataract was comparatively much low than Glaucoma among the study population and that was only 19%. Awareness of night blindness was reasonable in the study population but knowledge was moderate but not so good. A majority (55%) of the subjects reported other causes as common causes of night blindness even though the question asked was specifically about night blindness in childhood, which is most commonly due to vitamin A deficiency in the developing world (KP *et al*., 1987).

Awareness and knowledge of night blindness during childhood is important since vitamin A deficiency is common and is also associated with higher mortality in children. Vitamin A deficiency is reported to be largely confined to impoverished countries, neighborhoods and families. Knowledge about the interaction between diabetes and eye disease (40%) was very

poor among the study population. A study report in Australia shows that there is a large gap in the public's knowledge and understanding of eye disease that will need to be understood for eye health promotion activities (Livingston *et al*., 1998).

Fill the gap between the public's knowledge and understanding of eye disease for increasing of awareness among the population of Bangladesh is a must. According to this study a large portion of the study population (77%) were not concern about their family' present eye health condition. 82% of them never had an eye test. The percentage of the frequency of taking nutritional food everyday was only 17% and 32% were not concern about it for their optimum eye health. Hypertension control was significantly lower among younger than middle-aged individuals and older adults in all over the world (Brent *et al*., 2010), Where in Bangladesh only 36% of the study population were maintaining their blood pressure and obesity. Proper knowledge and education can increase the awareness of eye disease among the population. Awareness and knowledge regarding eye disease is still grossly inadequate in Bangladesh. Massive eye education programmes are urgently needed both in urban and rural area of Bangladesh.

5.2 Conclusion

In conclusion, the result of this survey based study suggests that there is an urgent need for health education in the study population in order to increase their level of awareness and knowledge about common eye diseases. This is particularly important in a developing country such as Bangladesh, with considerable investment in tertiary eye care. Based on the report's findings we would expect that awareness and knowledge of eye diseases would be worse in total population of Bangladesh. Increasing the awareness and knowledge of common eye diseases could lead to an increase in understanding and acceptance of the importance of routine eye examination for early detection and treatment of such conditions, thereby reducing visual impairment and cost of eye care. These data could help to develop effective health education and information programmes to reduce visual impairment among the population of Bangladesh.

Chapter six

References

References

Akbari, M.; Akbari, S.; Pasquale, L. R. (2009). *"The Association of Primary Open-angle Glaucoma with Mortality: A Meta-analysis of Observational Studies".* Archives of Ophthalmology 127 (2): 204–210.

Alice T., Philip Wang, Louis R. (2000), *"Determinants of glaucoma awareness in a general eye clinic".* Volume 107, Issue 2, Pages 303–308.

Alguire P (1990). *"The Eye Chapter 118 Tonometry of Basic Science".* London: Butterworth.

Allen D, Vasavada A (2006). *"Cataract and surgery for cataract".* BMJ 333 (7559): 128–32.

Allday E. (2012) *"Macular degeneration a growing problem".* Available at : http://www.sfgate.com/health/article/Macular-degeneration-a-growing-problem-3822617.php

American Journal of Ophthalmology (2001) *"the age-related eye disease study system for classifying age-related macular degeneration from stereoscopic color fundus photographs".* The age-related eye disease study report, Volume 132, Issue 5, Pages 668–681.

Anisimova SY, Anisimova SI, Larionov EV. (2012) *"Biological drainage – Xenoplast in glaucoma surgery (experimental and 10-year of clinical follow-up)"* , EGS Congress, Copenhagen. Retrieved, 2013.

Arthur J. Sit, MD (2006). *"Many types of glaucoma, one kind of damage to optic nerve". Chicago Tribune.* Retrieved 2015.

Banerjee Dr. (2006). *"A review and clinical evaluation of per-operative and post-operative complications in case of manual small incision cataract surgery and extracapsular cataract extraction with posterior chamber intra-ocular lens implantation"* . Retrieved 2014.

Best G, Amberger R, Baddeley D, Ach T, Dithmar S, Heintzmann R and Cremer C (2011). *"Structured illumination microscopy of autofluorescent aggregations in human tissue".* Micron, 42, 330-335

Bill S, Gilbert C, Sabur M A. (1997) *"Evaluation report on Modular Eye Care Projects*", West Sussex.

Black N, Browne J, van der Meulen J, Jamieson L, Copley L, Lewsey J (2008). *"Is there overutilisation of cataract surgery in England?".* British Journal of Ophthalmology 93 (1): 13–17.

Bollinger KE, Langston RH (2008). *"What can patients expect from cataract surgery?"*. Cleveland Clinic journal of medicine 75 (3): 193–196, 199–196.

Brent M. Egan, MD; Zhao Y, (2010) *"US Trends in Prevalence, Awareness, Treatment, and Control of Hypertension".*

Bulletin of the World Health Organization, (2001), vol.79, n.2, pp. 96-102.

Chang B, Smith RS, Peters M et al. (2001). *"Haploinsufficient Bmp4 ocular phenotypes include anterior segment dysgenesis with elevated intraocular pressure"*. BMC Genet. 2: 18.

Christen WG, Manson JE, Seddon JM, Glynn RJ, Buring JE, Rosner B, Hennekens CH (1992). *"A prospective study of cigarette smoking and risk of cataract in men".* JAMA 268 (8): 989–93.

Chew, MD; Traci E. Clemons, PhD; Matthew D. Davis, MD; Frederick L. Ferris III, MD; Gary R. Gensler, MS; Natalie Kurinij, PhD; Anne S. Lindblad, PhD; Roy C. Milton, PhD; Johanna M. Seddon, MD; and Robert D. Sperduto, MD (2007). *"The Relationship of Dietary Lipid Intake and Age-Related Macular Degeneration in a Case-Control Study"*. Archives of Ophthalmology.

Dasari B, Prasanthi JR, Marwarha G,Singh BB, Ghribi O. (2011). *"Cholesterol-enriched diet causes age-related macular degeneration-like pathology in rabbit retina"*. BMC Ophthalmol. 11: 22.

De Jong pt. (2006). *"Age-related macular degeneration".* N Engl J Med. 355 (14): 1474–1485. Dandona R. Prasad L.(1999), *"Utilization of eye care services in an urban population in southern India: the Andhra Pradesh eye disease study Banjara Hills*", Br J Ophthalmol 2000;84:22-27.

Deokule S. Sadiq S., Shah S. (2003), *"Chronic open angle glaucoma: patient awareness of the nature of the disease, topical medication, compliance and the prevalence of systemic symptoms".*

Emmett T. Cunningham, Paul Riordan-Eva. Vaughan & Asbury's general ophthalmology. (18th ed.). McGraw-Hill Medical.

Evans JR; Evans, Jennifer R (2006). *"Antioxidant vitamin and mineral supplements for slowing the progression of age-related macular degeneration".* Cochrane Database Syst Rev (2): CD000254.

Expert Committee on the Diagnosis and Classification of Diabetes Mellitus (2003). "Report of the expert committee on the diagnosis and classification of diabetes mellitus". Diabetes Care 26 (Suppl 1): S5–20.

Foster A. (1999) Sixth General Assembly IAPB. Beijing, China.

Geiss L. Saaddine J, Boyle J (2004) *"The Evolving Diabetes Burden in the United States."* Annals of Internal Medicine, Web. 22 Apr. 2014.

Greiner JV, Chylack LT (1979). *"Posterior subcapsular cataracts: histopathologic study of steroid-associated cataracts"*. Arch Ophthalmol 97 (1): 135–44.

Hirschler, Ben (2008). "Gene discovery may help hunt for blindness cure". Reuters. Retrieved 2008.

Health Technology Assessment (2003) *"Clinical effectiveness and cost–utility of photodynamic therapy for wet age-related macular degeneration: a systematic review and economic evaluation".* Vol. 7: No. 9.

Indo-Asian news service (2012). *"IIT Kharagpur pioneers Software for Fast Diagnosis of Diabetic Retinopathy".* Biharprabha News.

Islam F. Chakrabarti R. Silvia Z. (2015) *"Factors Associated with Awareness, Attitudes and Practices Regarding Common Eye Diseases in the General Population in a Rural District in Bangladesh."*

James R. Gavin III, MD. Stern, MD, (1997). *"Report of the Expert Committee on the Diagnosis and Classification of Diabetes Mellitus"*. Diabetes Care 20 (7): 1183–1197.

Johnson, Chris A. Owens, D. A., (2003); "The use of a visual illusion to detect glaucoma, In Visual Perception". Washington, D.C.: The American Psychological Association.

Jonathan C. Horton (2008) *"Disorders of the Eye",* in Harrison's Principles of Internal Medicine, 16th ed. Ch. 25.

John S. (2013). *"Choice of cell source in cell based therapies for retinal damage due to age related macular degeneration (AMD): A review"*. Journal of Ophthalmology 2013: 1.

Kahn HA, Leibowitz HM, Ganley JP, Kini MM, Colton T, Nickerson RS, Dawber TR (1977). *"The Framingham Eye Study. I. Outline and major prevalence findings".*Am J Epidemiol 106 (1): 17–32.

Kertes PJ, Johnson TM, ed. (2007) *"Evidence Based Eye Care".* Philadelphia, PA: Lippincott Williams & Wilkins.

KP Jr, Sommer A. (1987) *"Delivery of oral doses of vitamin A to prevent vitamin A deficiency and nutritional blindness".* Nutrition Policy Discussion Paper no. 7. Geneva: ACC/SCN, 1987.

Leffler CT, Schwartz SG, Stackhouse R, Davenport B, Spetzler K (2013).*"Evolution and impact of eye and vision terms in written English"*. JAMA Ophthalmol 131 (12): 1625–31.

Leffler CT, Amini L (2007). *"Interpretation of uniocular and binocular trials of glaucoma medications: an observational case series"*. BMC Ophthalmol 7: 17.

Livingston P. McCarty C. Taylor H. (1998) *"Knowledge, attitudes, and self-care practices associated with age related eye disease in Australia"* Br J Ophthalmol 82:780-785.

Li, M; Wang, M; Guo, W; Wang, J; Sun, X (2011). *"The effect of caffeine on intraocular pressure: a systematic review and meta-analysis",* 249 (3): 435–42.

Lipman RM, Tripathi BJ, Tripathi RC (1988). *"Cataracts induced by microwave and ionizing radiation".* Surv Ophthalmol 33 (3): 200–10.

Li M, Tmaca-Sonmez P, Othman M, Branham KE, Khanna R, Wade MS (2006). *"CFH haplotypes without the Y402H coding variant show strong association with susceptibility to age-related macular degeneration"*. Nature Genetics 38 (9): 1049–54.

Mathew MC, Ervin AM, Tao J, Davis RM (2012). *"Antioxidant vitamin supplementation for preventing and slowing the progression of age-related cataract."*

Masharani, Umesh (2006). *"Diabetes Ocular complications"*. Chronic Complications of Diabetes. Armenian Medical Network.

Mi, Xue-Song; Yuan, Ti-Fei; So, Kwok-Fai (2014). *"The current research status of normal tension glaucoma"*. Clinical Interventions in Aging 9: 1563–71.

Mozaffarieh M, Flammer J (2007). *"Is there more to glaucoma treatment than lowering IOP?"*. Surv Ophthalmol 52 (Suppl 2): S174–9.

Muhit MA, Shahjahan M, Hassan A, Wazed A, Ahmed N (2011) *"Parental knowledge, attitude and practice related to blindness of children in some selected Upazilla of Bangladesh."* 20(4):671-679.

National Institute of Ophthalmology (1999), National Workshop, Dhaka, Bangladesh.

National Center for Chronic Disease Prevention and Health Promotion, (2013), Division of Diabetes Translation, Available at: http://www.cdc.gov/chronicdisease/overview, last updated: August 26, 2015.

National Health Service England (2009) *"Central Mersey Diabetic Retinopathy Screening Programme",* England.

Neale RE, Purdie JL, Hirst LW, Green AC (2003). *"Sun exposure as a risk factor for nuclear cataract".* Epidemiology 14 (6): 707–712.

Olsen T, Jeppesen P (2012). *"The Incidence of Retinal Detachment after Cataract Surgery".* The Open Ophthalmology Journal 6: 79–82.

Osborne NN, Wood JP, Chidlow G, Bae JH, Melena J, Nash MS (1999). *"Ganglion cell death in glaucoma: what do we really know?"*. Br J Ophthalmol 83(8): 980–6.

O'Malley, PG (2012). *"Comparative effectiveness of anti-growth factor therapies for diabetic macular edema: summary of primary findings and conclusions".* Archives of internal medicine 172 (13): 1014–5.

Parikh RS, Parikh SR, Navin S, Arun E, Thomas R (2008). *"Practical approach to medical management of glaucoma".* Indian J Ophthalmol 56 (3): 223–30.

Pardianto G. (2006) *"Some difficulties on Glaucoma".* Mimbar Ilmiah Oftalmologi Indonesia.;3: 49–52.

Pardianto G.(2005). *"Understanding diabetic retinopathy".* Mimbar Ilmiah Oftalmologi Indonesia 2: 65–6.

Qihua Le, **Xiaodong Z. Ling Ge**, **Liangcheng Wu**, **(**2012**)** *"Impact of Dry Eye Syndrome on Vision-Related Quality of Life in a Non-Clinic-Based General Population".*

Quigley, H A; Broman, AT (2006). *"The number of people with glaucoma worldwide in 2010 and 2020".* British Journal of Ophthalmology 90 (3): 262–267.

Rahman, M. and Rahman, N. (2004) *"the prevalence of glaucoma in Bangladesh"*, *Dhaka.*

Rao, GN; Khanna, R; Payal, A (2011). *"The global burden of cataract."* Current opinion in ophthalmology 22 (1): 4–9.

Rajesh K. John, Catherine A. McCarty, & Gullapalli N. (2001) *"Awareness of eye diseases in an urban population in southern India."* Bull World Health Organ vol.79 n.2 Genebra.

Ramulu, Pradeep (2009). *"Glaucoma and disability: which tasks are affected, and at what stage of disease?".* Current opinion in ophthalmology 20 (2): 92–8.

Reddy SC (1999). *"Electric cataract: a case report and review of the literature."* European journal of ophthalmology 9 (2): 134–8.

Rhee, Douglas J. (2013). *"The Merck Manual Home Health Handbook".* Retrieved December 12, 2013.

Rosentreter, Andre; Schild AM; Jordan JF; Krieglstein GK; Dietlein TS (2010). *"A prospective randomised trial of trabeculectomy using mitomycin C vs an ologen implant in open angle glaucoma".* Eye 24 (9): 1449–57.

Roberts S, Woods C. (2008) *"Effects of a novel porous implant in refractory glaucoma".* ACVO abstract, Boston, MA.

Royal national institute of blind people,(2002) *"Posterior capsule opacification - why laser treatment is sometimes needed following cataract surgery"*, Available at : http://www.rnib.org.uk/eye-health-eye-conditions-z-eye-conditions/cataracts-laser-treatment-following-cataract-surgery.

Roberts, DL (2006). *"The First Year--Age Related Macular Degeneration".* (Marlowe & Company): 100.

Roberts, (2005). *"Artificial Lighting and the Blue Light Hazard".* Macular Degeneration Support Online Library.

Ryeom, Sandra; Folkman, Judah (2009). *"Role of Endogenous Angiogenesis Inhibitors in Down Syndrome".* Journal of Craniofacial Surgery 20 (Suppl 1): 595–6.

Sliney DH (1994*). "UV radiation ocular exposure dosimetry".* Doc Ophthalmol 88(3-4): 243–54.

Sommer A, Tielsch JM, Katz J. (1991). *"Relationship between intraocular pressure and primary open angle glaucoma among white and black Americans. The Baltimore Eye Survey".* Arch Ophthalmol. 109 (8): 1090–5.

Sperduto RD, Seigel D (1980). *"Senile lens and senile macular changes in a population-based sample".* Am J Ophthalmol 90 (1): 86–91.

Srinivas M. Mandal P. Rajesh K John.(2000) *"Open-angle glaucoma in an urban population in southern India".*

Tan JS, Wang JJ, Flood V, Rochtchina E, Smith W, Mitchell P. (2008). *"Dietary antioxidants and the long-term incidence of age-related macular degeneration: the Blue Mountain Eye Study".* Ophthalmology. 115 (2): 334–41.

The Economist (2015) *"Bionic eyes: A new device may restore vision to those whose sight is dwindling".*

Tsai JC, Song BJ, Wu L, Forbes M (2007). *"Erythropoietin: a candidate neuro protective agent in the treatment of glaucoma".* J Glaucoma 16 (6): 567–71.

Toke, Bek; Hammes, H-P; Porta, M. (2010). *"Experimental Approaches to Diabetic Retinopathy - Front Diabetes".* Clinical Presentations and Pathological Correlates of Retinopathy,(Basel). pp. 1–19.

Toh T, Morton J, Coxon J, Elder MJ (2007). *"Medical treatment of cataract". Clin. Experiment. Ophthalmol.* 35 (7): 664–71.

United States National Library of Medicine *(2009) "Causes and Risk Factors" of Diabetic Retinopathy.*

Van den Brûle J, Degueldre F, Galand A (1998). *"Cataractes incitées de médicament".* Revue médicale de Liège (in French) 53 (12): 766–9.

Wang N, Wu H, Fan Z. (2002). "Primary angle closure glaucoma in Chinese and Western populations". Chin Med J. 115 (11): 1706–15.

Williams DL, Munday P (2006). *"The effect of a topical antioxidant formulation including N-acetyl carnosine on canine cataract: a preliminary study".* Vet Ophthalmol 9 (5): 311–6.

Wong TY; Liew G; Tapp RJ. (2008*). "The Relationship of Fasting Glucose to Retinopathy: Re-visiting a Key Criterion Used to Diagnose Diabetes"*. Lancet 371 (9614): 736–43.

World Health Organization (2001) *"Awareness of eye diseases in an urban population in southern India."* vol.79 n.2.

World Health Organization, (Action plan 2006-2011), vision 2020 the right to sight.

World Health Organization, (2008). *"The global burden of disease"*. Geneva, Switzerland. p. 35.

Yanoff, Myron; Duker, Jay S. (2008). Ophthalmology (3rd ed.).

Zhou JB, Guan HJ, Qu J, Yang XJ, Peng D, Gu HY, (2008), *"A study on the awareness of cataract disease and treatment options in patients who need surgery in a rural area of Eastern China",* [18(4):544-550].